2

DOT/FAA/AR-95/5

Office of Aviation Research
Washington, D.C. 20591

Toxicity Assessment of Combustion Gases and Development of a Survival Model

Louise C. Speitel

Airport and Aircraft Safety
Research and Development Division
FAA Technical Center
Atlantic City International Airport, NJ 08405

July 1995

Final Report

This document is available to the public through the National Technical Information Service Springfield, Virginia 22161

U.S. Department of Transportation
Federal Aviation Administration

NOTICE

This document is disseminated under the sponsorship of the U.S. Department of Transportation in the interest of information exchange. The United States Government assumes no liability for the contents or use thereof. The United States Government does not endorse products or manufacturers. Trade or manufacturer's names appear herein solely because they are considered essential to the objective of this report.

Technical Report Documentation Page

1. Report No.	2. Government Accession No.	3. Recipient's Catalog No.
DOT/FAA/AR-95/5		

4. Title and Subtitle	5. Report Date
TOXICITY ASSESSMENT OF COMBUSTION GASES AND DEVELOPMENT OF A SURVIVAL MODEL	July 1995
	6. Performing Organization Code
7. Author(s)	8. Performing Organization Report No.
Louise C. Speitel	
9. Performing Organization Name and Address	10. Work Unit No. (TRAIS)
Airport and Aircraft Safety Research and Development Division FAA Technical Center Atlantic City International Airport, NJ 08405	11. Contract or Grant No.
12. Sponsoring Agency Name and Address	13. Type of Report and Period Covered
U.S. Department of Transportation Federal Aviation Administration Office of Aviation Research Washington, D.C. 20591	Final Report
	14. Sponsoring Agency Code

15. Supplementary Notes

16. Abstract

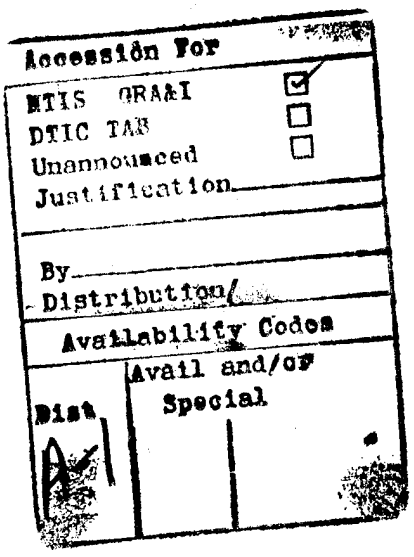
This report presents an extensive review of the literature on the toxic and thermal hazards relating to human survival in aircraft cabin fires. Studies by various authors of exposures to single and mixed gases on humans, primates, rats and mice are presented for different activity levels and a wide range of concentrations and temperatures. Regression equations giving the best fit were derived from these studies. The regression equation which was judged to best model the human escaping from an aircraft cabin fire was selected for each gas and utilized in the survival model. The effect of carbon dioxide increasing the uptake of other gases was included in the model.

This survival model uses incapacitation data to obtain a fractional effective dose for incapacitation (FED_I) and lethality data to obtain a fractional effective dose for lethality (FED_L). The time when either FED reaches 1 determines the exposure time available to escape from an aircraft cabin fire and to survive postexposure.

17. Key Words	18. Distribution Statement
Toxicity Review Toxicity Assessment Survival Model Fractional Effective Dose	This document is available to the public through the National Technical Information Service, Springfield, Virginia 22161.

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of Pages	22. Price
Unclassified	Unclassified	138	

TABLE OF CONTENTS



LIST OF ILLUSTRATIONS

LIST OF TABLES

LIST OF ABBREVIATIONS AND SYMBOLS

AMS	Acute Mountain Sickness
COHb	Carboxyhemoglobin
f	Respiratory Rate
FED_I	Fractional Effective Dose for Incapacitation
FED_L	Fractional Effective Dose for Lethality (LC_{50})
LC_{50}	Lethal Concentration - 50 Percent
MV	Minute Volume
P_B	Barometric Pressure
P_{N_2}	Partial Pressure of Nitrogen Gas
P_{O_2}	Partial Pressure of Oxygen Gas
RMV	Respiratory Minute Volume
S_aO_2	Oxygen Saturation
STPD	Standard Temperature and Pressure, Dry
t_{effect}	Time to Effect
$t_{exposure}$	Exposure Time
t_i	Time to Incapacitation
TLV	Threshold Limit Value
TUC	Time of Useful Consciousness
V_T	Tidal Volume

EXECUTIVE SUMMARY

This report presents an extensive review of literature on the toxic and thermal hazards relating to human survival in aircraft cabin fires. Studies by various authors of exposures to single and mixed gases on humans, primates, rats and mice at different activity levels are presented. Heat and oxygen depletion studies are included. Regression equations giving the best fit to the concentration time data were derived from those studies.

A new Combined Hazard Survival Model was created using selected regression equations to be used as a predictive tool to gauge human survivability in full-scale aircraft cabin fire tests. This new model uses incapacitation data to obtain a fractional effective dose for incapacitation (FED_I) and lethality data to obtain a fractional effective dose for lethality (FED_L). The time when either FED reaches 1 determines the exposure time available to escape from an aircraft cabin fire and survive postexposure.

LC_{50}s are the best measure of the ability to escape and survive for the irritant gases such as hydrogen fluoride (HF), hydrogen chloride (HCl), hydrogen bromide (HBr), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2). An LC_{50} is the concentration for a given exposure duration which results in death or subsequent death of 50 percent of the animals. Exposure to high concentrations of these gases may not prevent escape but may result in subsequent death due to respiratory system damage. The LC_{50}s are less than the incapacitation concentrations for these gases.

Time to incapacitation (t_i) is the best measure of the ability to escape and survive exposure to the narcotic gases such as carbon monoxide (CO), carbon dioxide (CO_2), hydrogen cyanide (HCN), and low oxygen. The incapacitation concentration is less than the LC_{50} for these gases. If a passenger were incapacitated, one can assume that subsequent death would occur by rapidly spreading fire or toxic gases. The FED_Is for each hazard are added to give the total FED_I as a function of time.

The effect of CO_2 on increasing the uptake of other gases was factored into the concentration term in the FED equation for all gases with the exception of CO_2 and oxygen. Higher respiratory minute volumes due to CO_2 exposure was found to be an important factor in predicting the time to incapacitation or death.

The new FED_I model was exercised for selected full-scale aircraft cabin fire tests conducted at the FAA Technical Center Full-Scale Fire Test Facility, where concentrations of toxic gases, smoke, and temperature are monitored at various locations in the fuselage as a function of time.

INTRODUCTION

OBJECTIVES.

The objectives of this paper are as follows: (1) Provide a literature review of the most recent relevant toxicity studies of single and mixed gases of major toxicants found in aircraft cabin fires, including heat and oxygen depletion studies; (2) Provide a literature review of resultant regression equations, based on experimental animal data, relating lethal and incapacitative exposure times to a wide range of gas concentrations and temperatures for a variety of species and activity levels; (3) Create a new, improved Combined Hazard Survival Model using selected regression equations for each hazard. This Combined Hazard Survival Model can be used as a predictive tool to gauge human survivability and the relative contribution of each hazard in full-scale aircraft cabin fire tests; and (4) Exercise and compare old and new survival models for selected full-scale aircraft cabin fire tests to predict time to incapacitation.

BACKGROUND.

The following question occurs: Which measure of toxicity is more meaningful for survival in aircraft fires: time to incapacitation (t_i) or lethal concentration-50 percent (LC_{50})? LC_{50}s include not only fatalities during exposure but also postexposure lethalities. Exposure to high concentrations of irritant gases such as HCl and acrolein may not prevent escape but may result in subsequent death due to respiratory system damage. For these irritant gases, LC_{50}s are a better measure of ability to survive exposure in the postexposure time period.

However, if the incapacitating concentration is less than LC_{50} for a particular gas exposure, t_i would determine the ability to escape and survive (e.g., gases producing narcosis: CO, CO_2, HCN, and low O_2). If a passenger were incapacitated by toxic gases and other hazards, it is safe to assume that subsequent death would occur by rapidly spreading fire or toxic gases. For these narcotic gas hazards, t_i is the better measure of ability to escape an aircraft fire.

Thus, a survival model must both predict the ability to escape and the ability to survive after evacuation. The survival model developed in this report uses t_i values to determine the ability to escape and LC_{50} values to determine the ability to survive postexposure. This survival model includes the effect of heat and oxygen depletion.

DISCUSSION - LITERATURE REVIEW OF HAZARDS

INDIVIDUAL GASES AND HEAT.

CARBON MONOXIDE.

Incapacitation. Purser[1] has conducted experiments on monkeys illustrating the nature of the decline in behavioral task performance with exposure to CO. During the early stages of exposure, as the carboxyhemoglobin concentration builds up gradually in the blood, the effects are minimal. In active animals, the first minor signs of behavioral deficits did not occur until concentrations of 15% to 20% carboxyhemoglobin (COHb) were reached. Purser next observed a sudden rapid decline in behavioral task performance accompanied briefly by signs similar to severe alcohol intoxication which led rapidly to a state of deep coma. Purser's experiments on monkeys showed that the onset of significant effects is sudden (decreased respiration, severe decrease in heart rate, and greatly increased slow-wave activity indicative of cerebral depression). The degree of incapacitation rapidly becomes severe, so by the time a victim is aware he is affected, effective action is probably not possible.

Similar results have been obtained in humans. Stewart et al.[2] reported the first symptom (headache) to occur at 15% to 20% COHb.

Active subjects may be seriously affected by COHb concentrations that have only minor effects on sedentary subjects. A subject whose blood contains 30% carboxyhemoglobin (and 70% oxyhemoglobin, with a consequent reduction in oxygen-carrying capacity) may be able to function normally at rest. However, if he starts to perform light work, the increased oxygen extraction (increased fall of oxygen content as blood flows through the tissues) reduces the partial pressure of oxygen (P_{O_2}) of the blood at the venous ends of the tissue capillaries to a very low value. The consequent hypoxia causes collapse. The simple act of rising from a seated position can precipitate loss of consciousness.

The Stewart equation predicts the COHb concentration in the blood for short exposures (less than one hour) at high concentrations when the blood concentration is well below saturation level. This equation was derived from experimental human exposures by Stewart et al.[3].

$$\%\ COHb = (3.317 \times 10^{-5})\ (ppm\ CO)^{1.036}\ (RMV)\ (t) \qquad (1)$$

where

RMV = volume of air breathed per minute (L/min)

t = exposure time (min)

Figure 1, generated from standard reference data by Purser[1], shows a probable time to incapacitation for a 70 kg (154 lb) human exposed to different CO concentrations at 3 levels of activity. The figure shows that the level of activity can have a major effect on time to incapacitation and the COHb concentration at incapacitation.

Using curve B (figure 1) for light work, walking at 6.7 km/hr (3.98 mi/hr) and an RMV of 25 liters/minute and substituting these values into equation (1), Purser obtained an equation for t_i:

$$t_i = \frac{30}{8.2925 \times 10^{-4} \times C_{CO}^{1.036}} = \frac{36{,}177}{C_{CO}^{1.036}}$$ where C_{CO} is expressed in parts per million CO (2)

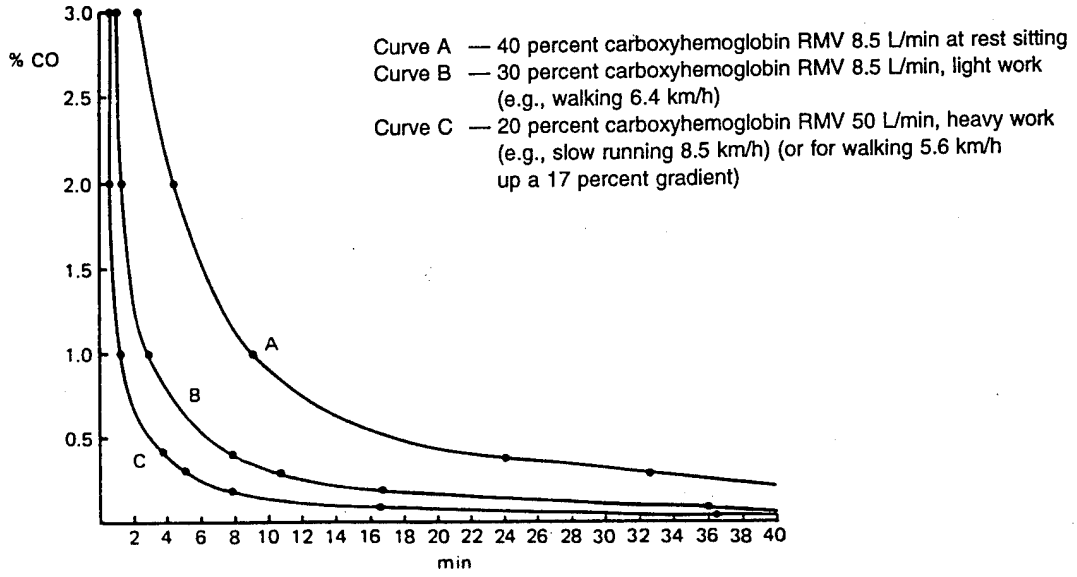


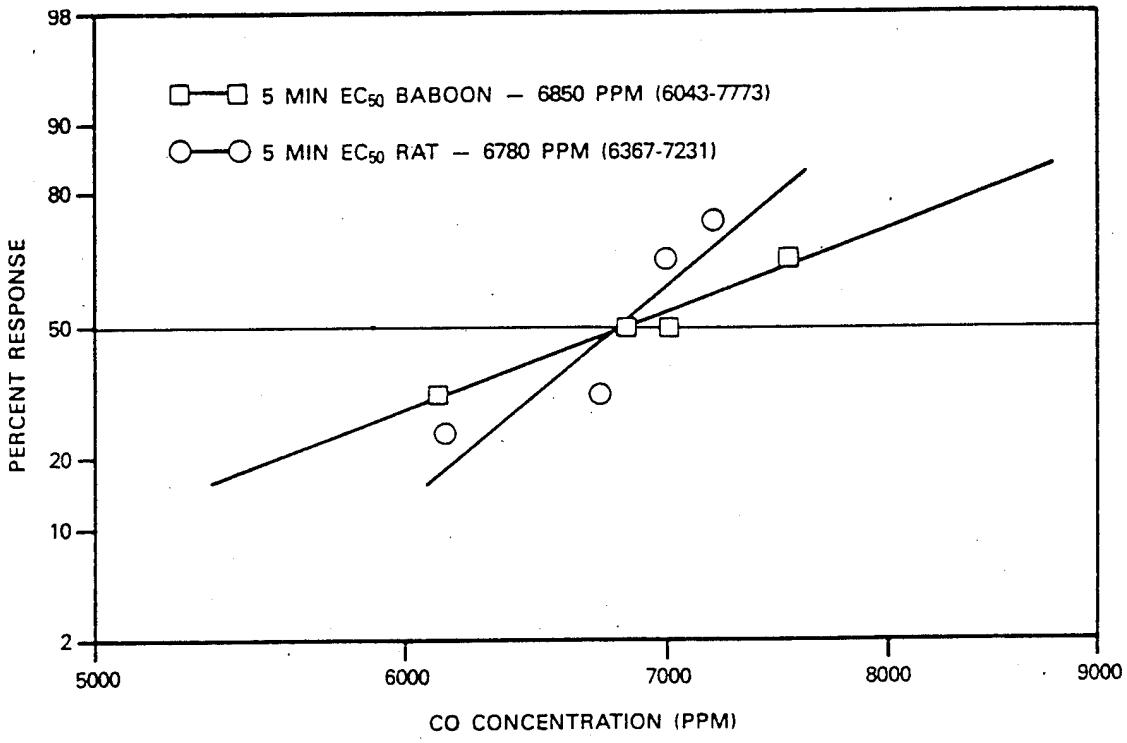
FIGURE 1. CARBON MONOXIDE TIME TO INCAPACITATION FOR A 70 kg HUMAN AT DIFFERENT ACTIVITY LEVELS

Exposures of men to 100 ppm CO for 3 hours produces no effect, but a nine-hour exposure causes headache and nausea according to Henderson and Haggard[4].

Kaplan et al. developed an avoidance task for use with the juvenile African Savannah baboon. After a 5-minute exposure, the animal was required to select and depress the correct lever to open an escape door and then to exit into the adjacent compartment of a shuttlebox. The gas concentration at which 50% of the animals could escape (the EC_{50}) was 6850 ppm for a 5-minute exposure (Ct product = 34,250 ppm•min).

$$t_i = \frac{34{,}250}{C_{CO}} \quad (3)$$

With a comparable shuttlebox and escape paradigm for rats, the EC_{50} of CO was 6780 ppm for a 5-minute exposure (Ct product = 33,900 ppm•min) (figure 2)[5]. This indicates that the rat may be an appropriate model for predicting the incapacitating effect of CO exposure in primates.



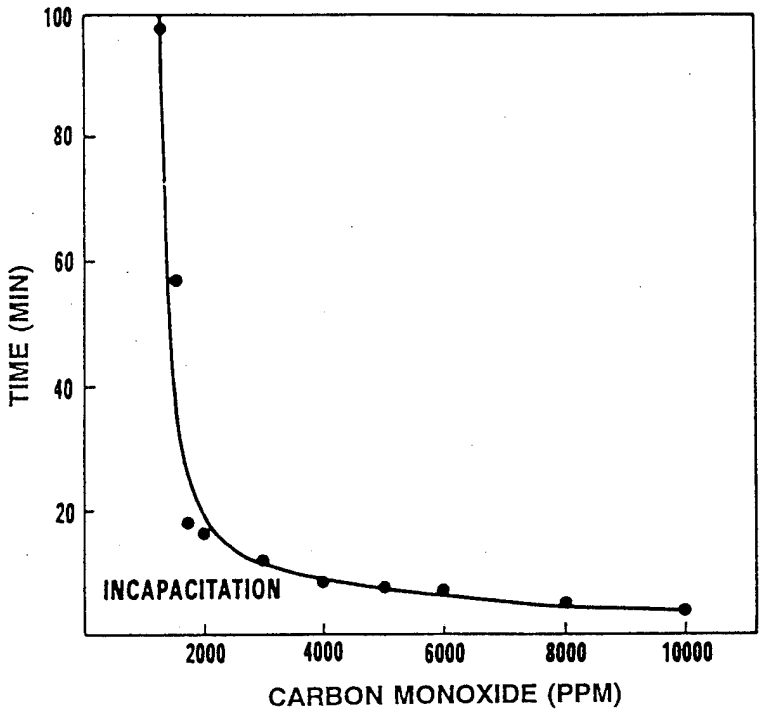
Reprinted with permission from the Journal of Fire Sciences, Copyright 1985 by Technomic Publishing Co., Inc.[5]

FIGURE 2. EFFECTS OF CARBON MONOXIDE ON ESCAPE IMPAIRMENT IN RATS AND JUVENILE BABOONS (KAPLAN)

Hartzell et al. exposed 148 rats, 6 per experiment, to various CO concentrations using the leg-flexion shock avoidance technique. The rat is positioned in a tubular restrainer such that an electric shock is delivered to one hind leg upon contact with a suspended metal platform. Upon exposure to a toxicant atmosphere, incapacitation is said to occur when the rat is unable to avoid the shock. Exposure concentrations ranged from 1300 to 10,000 ppm[6]. Concentrations of 1300 and 1500 ppm resulted in standard deviations of t_i approaching the average t_i for each experiment. Discarding these data points and considering exposures from 1700 ppm (t_i = 18.1 min) to 10,000 ppm (t_i = 3.7 min), the average Ct product resulting in incapacitation is 37,300 ppm•min (figures 3 and 4). Hartzell obtained a regression equation for his data.

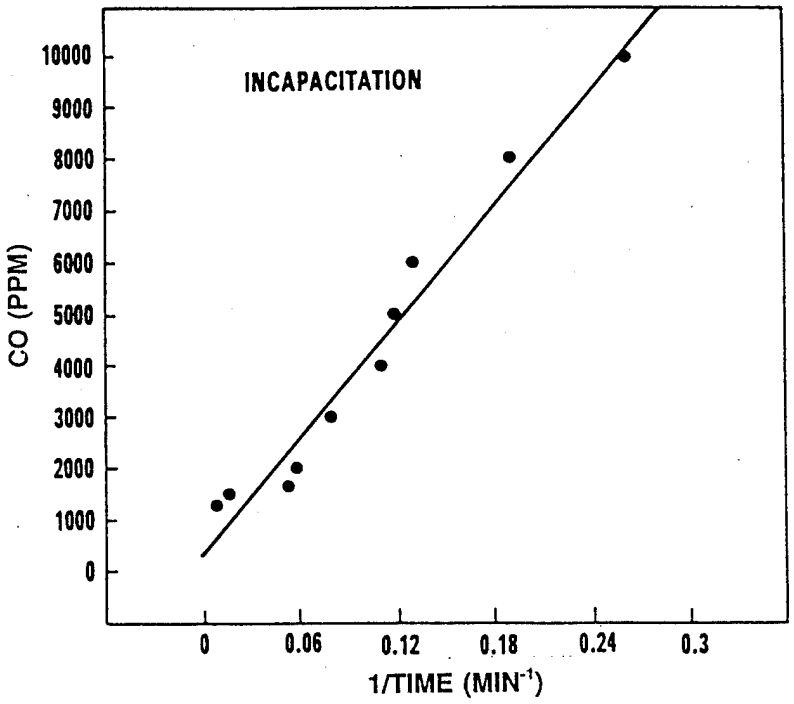
$$t_{i\,CO} = \frac{36{,}509}{C_{CO} - 233} \qquad \text{where } C_{CO} > 233 \text{ ppm} \qquad (4)$$

(*in minutes*)



Reprinted with permission from the Journal of Fire Sciences, Copyright 1985 by Technomic Publishing Co., Inc.[6]

FIGURE 3. TIME TO INCAPACITATION FOR RESTRAINED RATS EXPOSED TO CARBON MONOXIDE (HARTZELL)



Reprinted with permission from the Journal of Fire Sciences, Copyright 1985 by Technomic Publishing Co., Inc.[6]

FIGURE 4. $1/t_i$ FOR RESTRAINED RATS EXPOSED TO CARBON MONOXIDE (HARTZELL)

Crane et al. exposed 42 rats to various CO concentrations in an exposure chamber consisting of a motor-driven drum controlled at 6 cm/sec and a shock grid. If the subject animal failed to maintain the selected walking speed he was carried back onto the shock platform. Time to incapacitation was recorded when all walking motion ceased. Exposure concentrations ranged from 1437 ppm (t_i = 18.1 min) to 5992 ppm (t_i = 3.6 min) with an average Ct product of 28,395 ppm•min required for incapacitation (figure 5)[7]. Crane obtained the following regression equation for his data.

$$t_{i\,CO} = \frac{25{,}017}{C_{CO} - 225} + 0.3 \qquad \text{when } C_{CO} > 225 \text{ ppm} \qquad (5)$$

(in minutes)

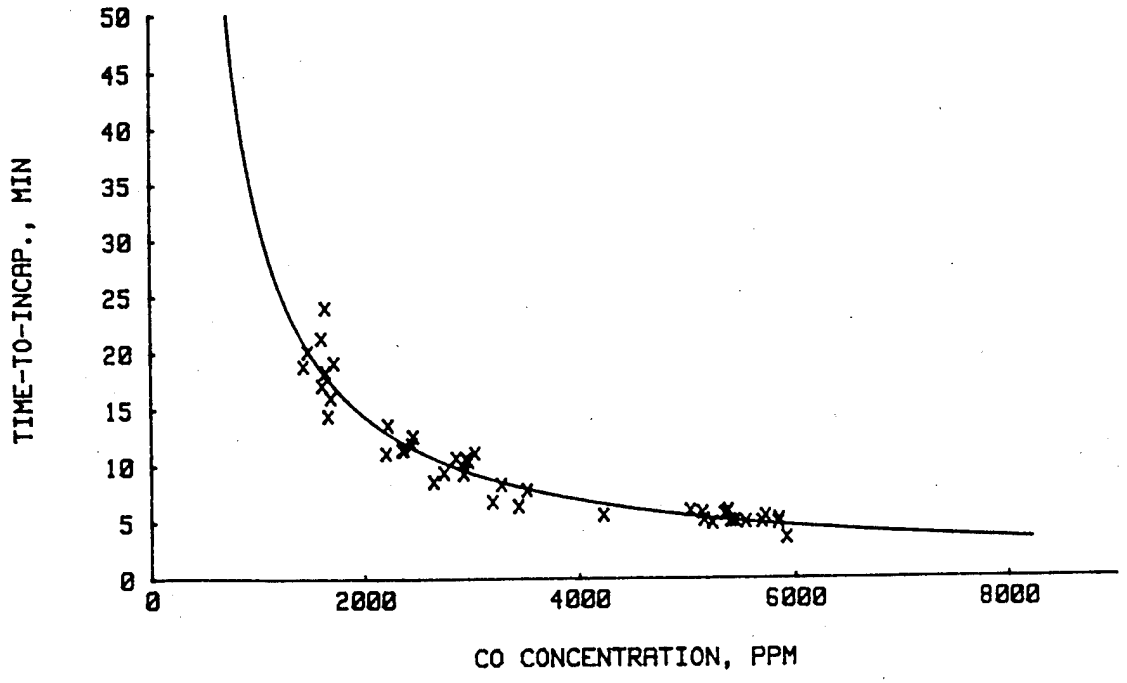
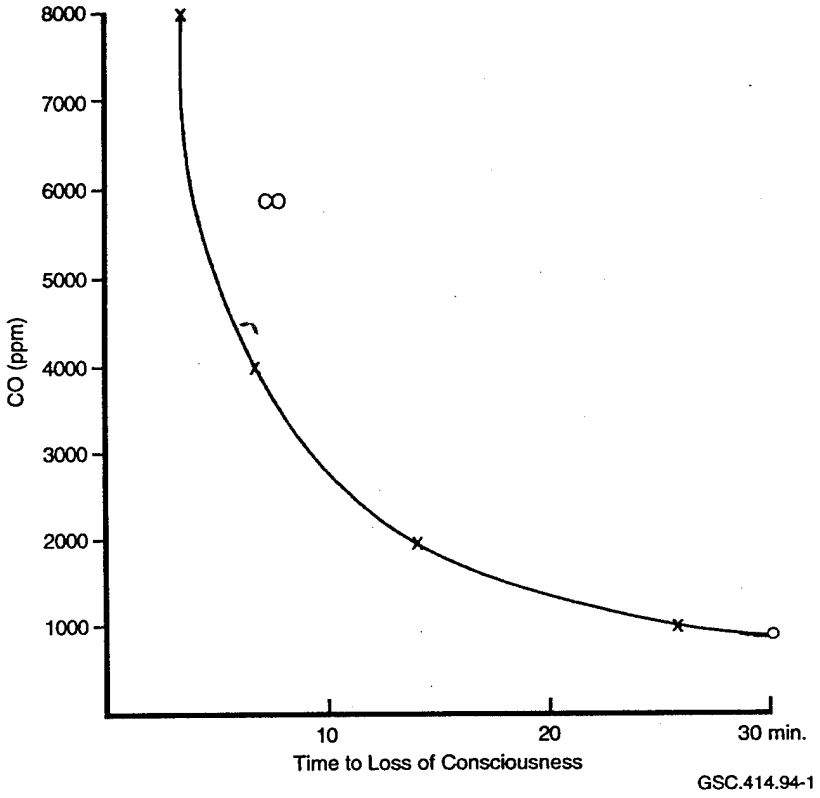


FIGURE 5. TIME TO INCAPACITATION FOR ACTIVE RATS EXPOSED TO CARBON MONOXIDE (CRANE)[7]

Purser developed a behavioral task involving trained monkeys moving bodily over a distance of 1 meter. He exposed active monkeys to four different CO concentrations. A CO concentration of 1000 ppm resulted in a Ct product of 26,600 ppm•min (at 2000 ppm Ct = 28,097 ppm•min; at 4000 ppm Ct = 26,868 ppm•min; at 8000 ppm Ct = 26,086 ppm•min)[1,8] (figure 6). Speitel averaged the Ct products of Purser's data:

$$t_{i\,CO} = \frac{26{,}912}{C_{CO}} \quad \text{(in minutes)} \tag{6}$$



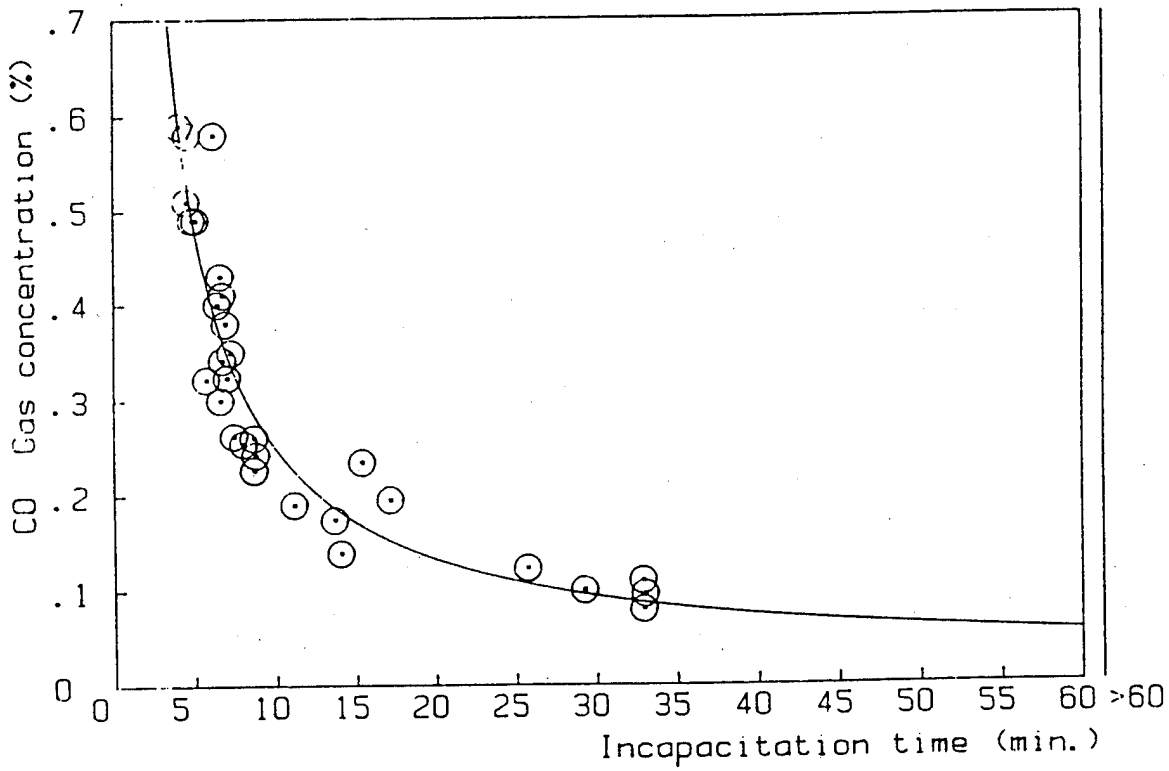
Reprinted with permission from the SFPE Handbook of Fire Protection Engineering, Copyright 1988 by Society of Fire Protection Engineers[1]

FIGURE 6. TIME TO INCAPACITATION FOR ACTIVE MONKEYS EXPOSED TO CARBON MONOXIDE (PURSER)

The preceding rat data indicates that the incapacitating Ct product appears to depend on the test method, i.e., whether the animal is active or restricted. The level of activity is the greatest for the rotating wheel/shock method (C•t = 28,395), followed by the escape task method (C•t = 33,900), followed by the leg flexion method where the rats are immobilized (C•t = 37,300).

Sakurai[9] exposed mice in motor-driven rotating wheels to various CO concentrations. Eight mice were used for each exposure concentration. He plotted gas concentration vs. time to incapacitation as illustrated in figure 7. He obtained a fit for this data with the following regression equation.

$$t_i = \frac{2.34}{C_{CO} - 0.016} \qquad \text{when } C_{CO} > 0.016\% \qquad (7)$$



Reprinted with permission from the Journal of Fire Sciences, Copyright 1989 by Technomic Publishing Co., Inc.[9]

FIGURE 7. TIME TO INCAPACITATION FOR ACTIVE MICE EXPOSED TO CARBON MONOXIDE (SAKURAI)

Lethality. Hartzell et al.[10] exposed 6 rats at a time to various CO concentrations for 30 minutes. The rats were restrained. Incapacitation was determined using conventional leg-flexion shock avoidance apparatus. The rats were monitored 14 days postexposure. The 30-minute LC_{50} was found to be 6415 ppm. The Ct product for lethality $L(Ct)_{50}$ is approximately 192,450 ppm•min. (See table 1.)

Levin et al.[11] calculated two sets of LC_{50} values with 95% confidence limits for CO in air for a series of exposure times ranging from one to 60 minutes. The LC_{50} values were calculated using deaths within exposure plus 24 hours. These LC_{50} values were obtained with rats supplied by the Harlan Sprague-Dawley Company (pre-1986, 30-minute values), Hilltop Lab Animal Company (all other pre-1986 values) or from Taconic Farms (1986 and later values) and are shown in table 2. Levin et al. determined that the LC_{50} values shifted when the animal supplier changed, even though both sets were designated Fisher 344 male rats. Therefore Levin notes it is important in any set of experiments to determine the LC_{50} values for the specific animals being used and to periodically check the values to ensure that they have not changed. The two sets of values are plotted in figure 8 along with the plot of the CO concentration-time profiles. The concentration-time products vary markedly for LC_{50} data with concentration.

TABLE 1. SUMMARY OF HARTZELL'S CARBON MONOXIDE LC_{50} DATA FOR RESTRAINED RATS

Average CO (ppm)	Number Incapacitation	Mean Time To Incapacitation (min)	Exposure Number Dead	Postexposure Number Dead	Total Number Dead
4176	5/6	13.173	0/6	0	0/6
5030	6/6	10.483	2/6	0	2/6
6017	6/6	6.65	3/6	0	3/6
7230	6/6	9.406	3/6	0	3/6
8633	6/6	4.106	5/6	0	5/6
10,951	6/6	2.833	6/6	0	6/6

TABLE 2. SUMMARY OF LEVIN'S CARBON MONOXIDE LC_{50} DATA FOR RESTRAINED RATS

Data Prior to 1986				1986 Data		
Exposure Time (min)	C_{CO} (ppm)		C_{CO}•t (ppm•min)	C_{CO} (ppm)		C_{CO}•t (ppm•min)
1	-----	----------	------	107,000	(102,000-112,000)	107,000
2	36,000	(34,000-38,000)	7200	42,500	(42,000-43,000)	85,000
5	12,600	(12,400-12,800)	63,000	14,000	(12,000-16,000)	70,000
10	7600	(7300-7900)	76,000	9800	(9200-10,500)	98,000
20	5600	(5400-5800)	112,000	7400	(7100-7500)	148,000
30	4600	(4400-4800)	138,000	6600	(6100-7300)	198,000
60	3400	(3100-3600)	204,000	4900	(4600-5200)	294,000

Note that Levin's 30-minute LC_{50} (1986 data) is in agreement with Hartzell's 30-minute LC_{50} of 6415 ppm. Speitel derived an equation to fit Levin's 1986 LC_{50} data.

$$t_{exposure\ CO} = \frac{58{,}000}{C_{CO} - 4000} + 0.4 \quad Linear\ fit\ for\ C_{CO} > 9000\ ppm \qquad (8)$$

$$(in\ minutes) = \exp\left[5.85 - (3.70 \times 10^{-4})\ (C_{CO})\right] \quad 2000\ ppm \le C_{CO} \le 9000\ ppm$$

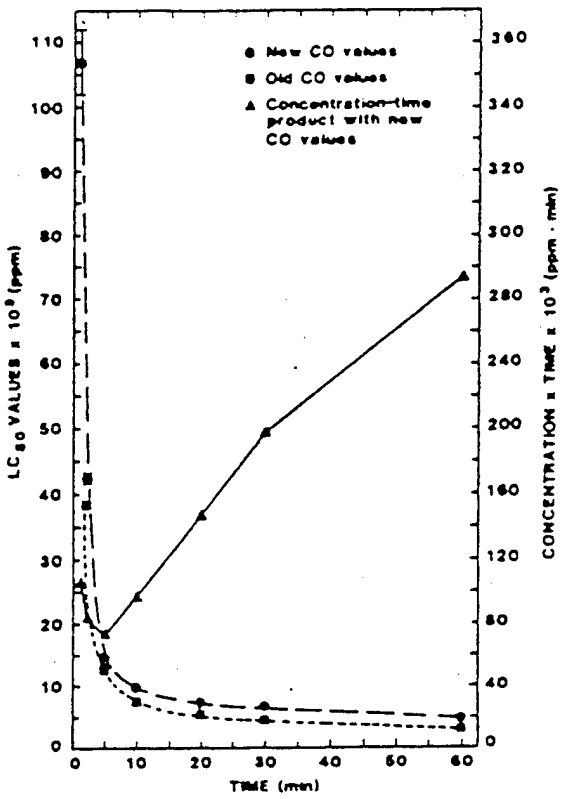


FIGURE 8. LC_{50}S AND CONCENTRATION-TIME PRODUCTS FOR RESTRAINED RATS EXPOSED TO CARBON MONOXIDE (LEVIN)[11]

HYDROGEN CYANIDE. HCN could be particularly dangerous in fires due to its rapid "knockdown" effect. Low HCN levels in the 100 and 200 ppm range could cause fire victims to lose consciousness rapidly and consequently to die later as a result of accumulation of CO or other hazards. For this reason, time to incapacitation is the endpoint of choice for HCN toxicity measurements in aircraft cabins.

Purser et al. conducted animal experiments[12,13] and found that at HCN concentrations below approximately 80 ppm the effects were minor over periods of up to 1 hour with mild background hyperventilation. At concentrations from 80 ppm to approximately 180 ppm, an episode of hyperventilation with subsequent unconsciousness occurred at some time during a 30-minute period; there was a loose linear relationship between HCN concentration and time to incapacitation. Above 180 ppm the hyperventilatory episode began immediately with unconsciousness occurring within a few minutes. Purser et al.[13] studied the effects of HCN in monkeys sitting in chairs, with gas administered by mask. Incapacitation was defined as a semiconscious state with loss of motor tone. The data are listed in table 3.

TABLE 3. SUMMARY OF PURSER'S HYDROGEN CYANIDE INCAPACITATION DATA FOR INACTIVE MONKEYS

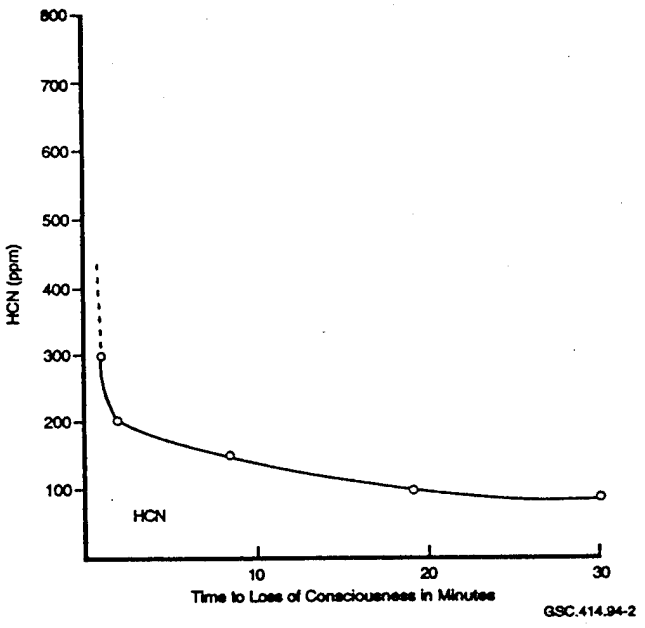
Concentration HCN (ppm)	Time to Incapacitation (min)
156	8
147	8
123	15
102	16
100	19

Purser plotted HCN concentration vs time to loss of consciousness based on various primate experiments he conducted over the years[1,14]. This is illustrated in figure 9. This curve has an almost linear portion resulting in a Ct product of approximately 2000 ppm•min at 100 ppm HCN and 400 ppm•min at 180 ppm[1]. This violates Haber's rule which predicts a constant Ct product. Haber recognized this deviation for HCN in 1924[15]. Figure 9 shows that outside the linear region, a small increase in HCN concentration causes a large decrease in time to incapacitation. Purser obtained the following equation to fit his data:

$$t_{iHCN} = \frac{185 - C_{HCN}}{4.4} \qquad \text{when } 80 \leq C_{HCN} \leq 180 \text{ ppm} \qquad (9)$$

(*in minutes*)

$$= \exp\,(5.396 - 0.023\ C_{HCN}) \qquad \text{when } C > 180 \text{ ppm}$$



Reprinted with permission from the SFPE Handbook of Fire Protection Engineering, Copyright 1988 by The Society of Fire Protection Engineers[1]

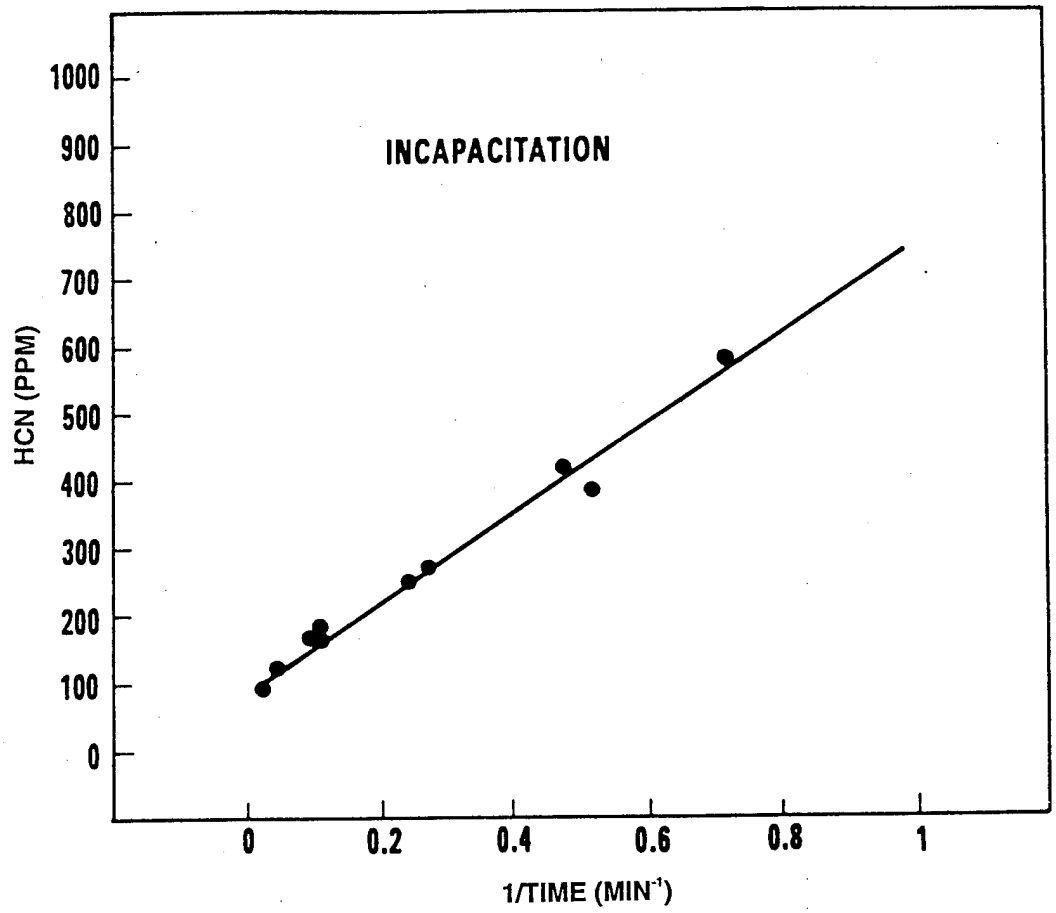
FIGURE 9. TIME TO INCAPACITATION FOR SEATED MONKEYS EXPOSED TO HYDROGEN CYANIDE (PURSER)

Data on human exposures to HCN are limited, but Kimmerle[16] does quote some approximate data on HCN lethality. He reports that 50 ppm HCN may be tolerated by man for 30 to 60 minutes, 100 ppm may be fatal after 1 hour, 135 ppm may be fatal after 30 minutes, 181 ppm may be fatal after 10 minutes, and 280 ppm may be immediately fatal. Data on human incapacitation, however, is not available.

Hartzell et al. exposed 72 rats, 6 per experiment, to various HCN concentrations using the leg-flexion shock avoidance technique. Exposure concentrations ranged from 95 ppm (43.8 minute mean t_i, C•t = 4161 ppm•min) to 592 ppm (1.4 minute mean t_i, C•t = 829 ppm•min). The Ct products decreased as the concentrations increased (figure 10)[6]. Hartzell obtained the following regression equation for his data:

$$t_{iHCN} = \frac{698}{C_{HCN} - 92} \qquad \text{when } C_{HCN} > 92 \text{ ppm} \qquad (10)$$

(in minutes)



Reprinted with permission from the Journal of Fire Sciences, Copyright 1985 by Technomic Publishing Co., Inc.[6]

FIGURE 10. $1/t_i$ FOR RESTRAINED RATS EXPOSED TO HYDROGEN CYANIDE (HARTZELL)

Crane et al. exposed 30 rats to various HCN exposures in an exposure chamber consisting of a motor-driven drum controlled at 6 cm/sec and a shock grid. If the subject animal failed to maintain the selected walking speed he was carried back onto the shock platform. Time to incapacitation was recorded when all walking motion ceased. Exposure concentrations ranged from 75 ppm (27.9 minute mean t_i, C•t = 1642 ppm•min) to 273 ppm (2.8 minute mean t_i, C•t = 764 ppm•min). The Ct product decreased as HCN concentrations increased. The data along with the best fit curve to the data are illustrated in figure 11[7]. Crane obtained the following equation to fit his data.

$$t_{iHCN} = \frac{564}{C_{HCN} - 63} \qquad \text{when } C_{HCN} > 63 \text{ ppm} \qquad (11)$$

(in minutes)

The incapacitating Ct product for rats appears to depend on the test method, i.e., whether the animal is active or restrained. The level of activity is the greatest for the rotating drum/shock method, (C•t = 734 ppm•min, C_{HCN} = 216 ppm, t_i = 3.4 minutes) followed by the leg-flexion method where the rats are immobilized (C•t = 1691 ppm•min, C_{HCN} = 190 ppm, t_i = 8.9 minutes).

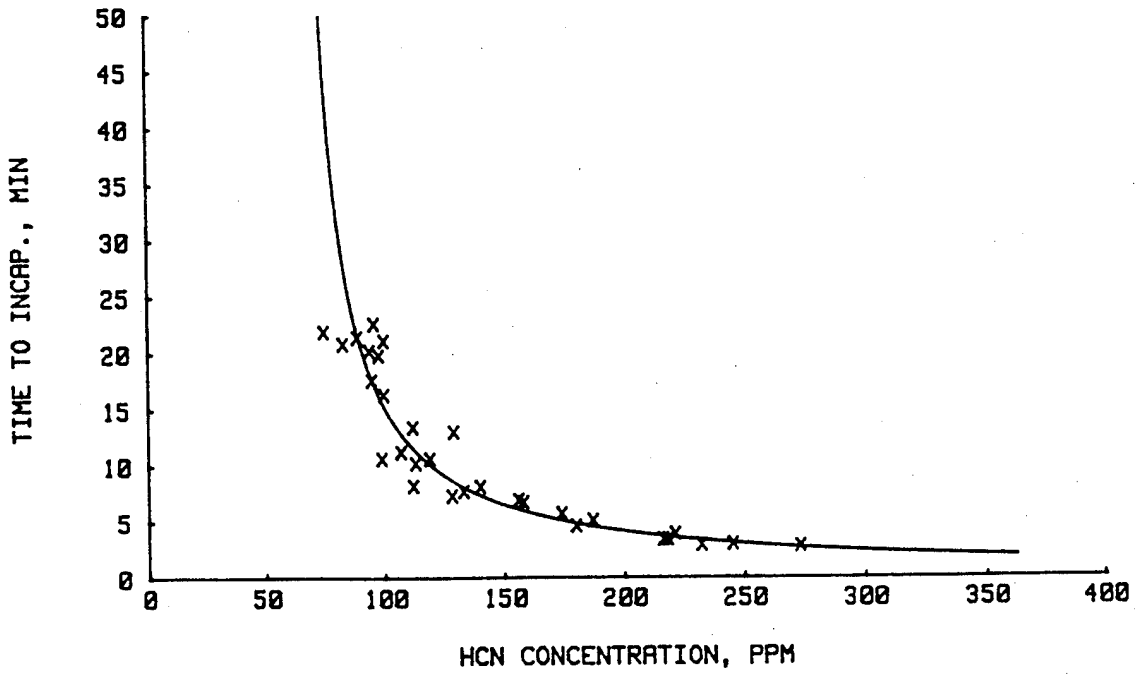
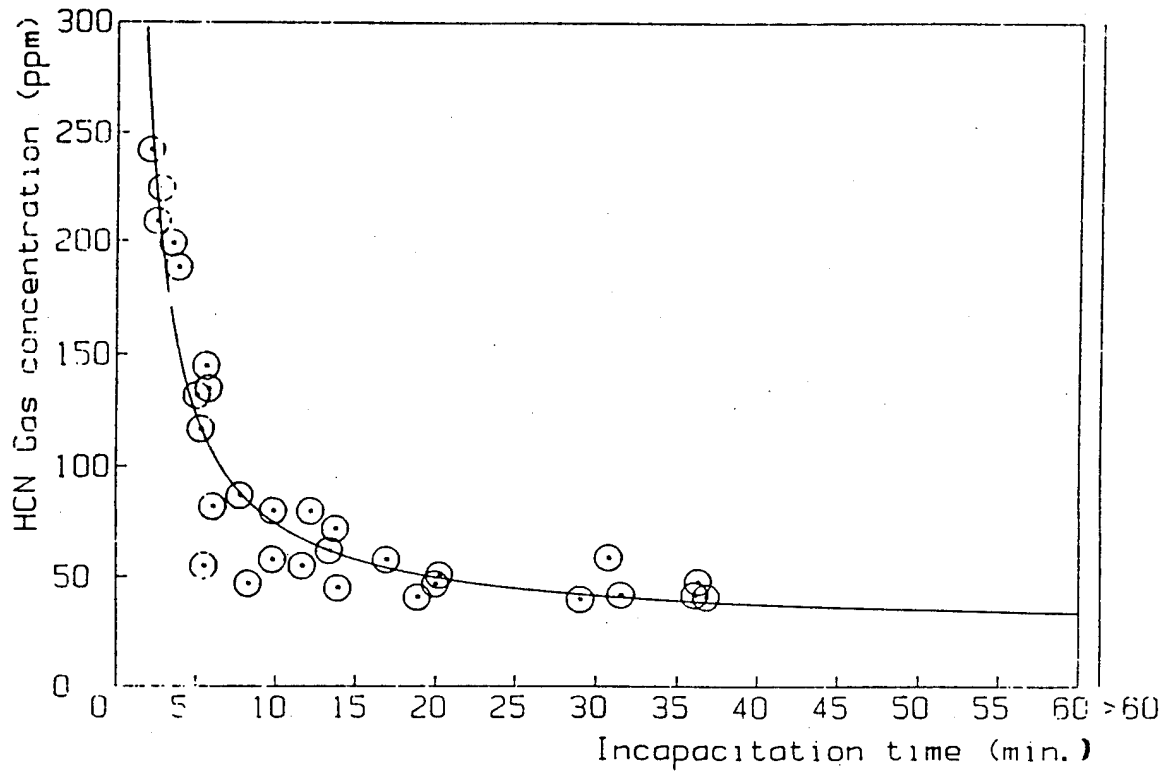


FIGURE 11. TIME TO INCAPACITATION FOR ACTIVE RATS EXPOSED TO HYDROGEN CYANIDE (CRANE)[7]

Sakurai[9] exposed mice in motor-driven rotating wheels to various HCN concentrations. Eight mice were used for each concentration. He plotted gas concentration vs. time to incapacitation as illustrated in figure 12 and obtained a best fit regression equation to fit this data:

$$t_i = \frac{491}{C_{HCN} - 25.3} \qquad \text{when } C_{HCN} > 25.3 \text{ ppm} \qquad (12)$$



Reprinted with permission from the Journal of Fire Sciences, Copyright 1989 by Technomic Publishing Co., Inc.[9]

FIGURE 12. TIME TO INCAPACITATION FOR ACTIVE MICE EXPOSED TO HYDROGEN CYANIDE (SAKURAI)

Levin et al. obtained the LC_{50} and EC_{50} values for Fischer 344 rats which were calculated for HCN for 1-, 2-, 5-, 10-, 20-, 30-, and 60-minute exposures plus relevant postexposure periods using the NBS Toxicity Test Method Apparatus. The LC_{50}s are based on deaths occurring within exposure plus 24 hours. The EC_{50}s are based on the lack of a righting reflex immediately following exposure. The LC_{50} values obtained and their 95% confidence limits are shown in table 4[11].

It can be noted from this table that the $LC_{50}t$ product is constant for this data up to 30 minutes. The average $L(Ct)_{50}$ product for these exposures is 3108 ppm•min. Speitel obtained a best fit regression equation to fit Levin's LC_{50} data. This best fit is illustrated in figure 13.

$$t_{exposure} = \frac{2586}{C_{HCN} - 43.2} \qquad \text{when } C_{HCN} > 43.2 \text{ ppm} \qquad (13)$$

TABLE 4. ACUTE INHALATION EXPOSURES OF RATS TO HYDROGEN CYANIDE (LEVIN)

Exposure time (min)	Lethality LC_{50} (ppm)	$L(Ct)_{50}$ (ppm•min)	Incapacitation EC_{50} (ppm)
1	3000 (2500-3600)	3000	1700 (1200-2400)
2	1600 (1400-1800)	3200	1100 (800-1300)
5	570 (460-710)	2850	390 (310-490)
10	290 (250-340)	2900	170 (150-190)
20	170 (160-180)	3400	Not Determined
30	110 (95-130)	3300	Not Determined
60	90	5400	Not Determined

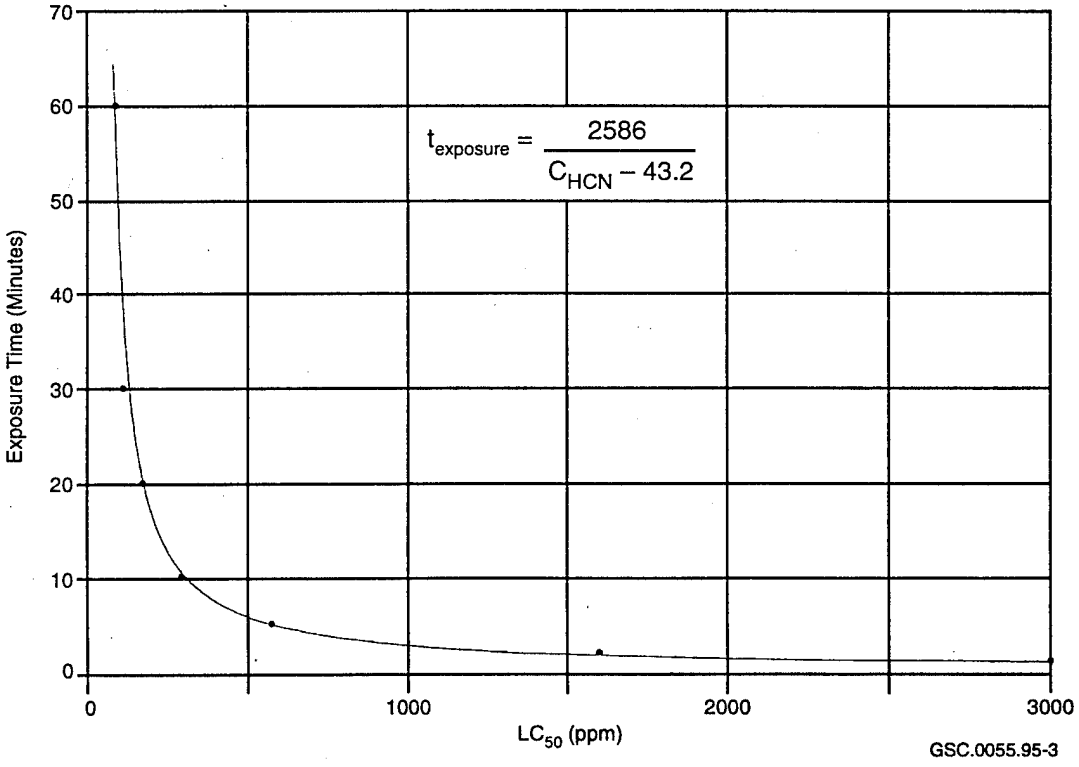


FIGURE 13. HYDROGEN CYANIDE LC_{50} VALUES FOR DIFFERENT EXPOSURE TIMES PLUS A 24-HOUR POSTEXPOSURE PERIOD (LEVIN)

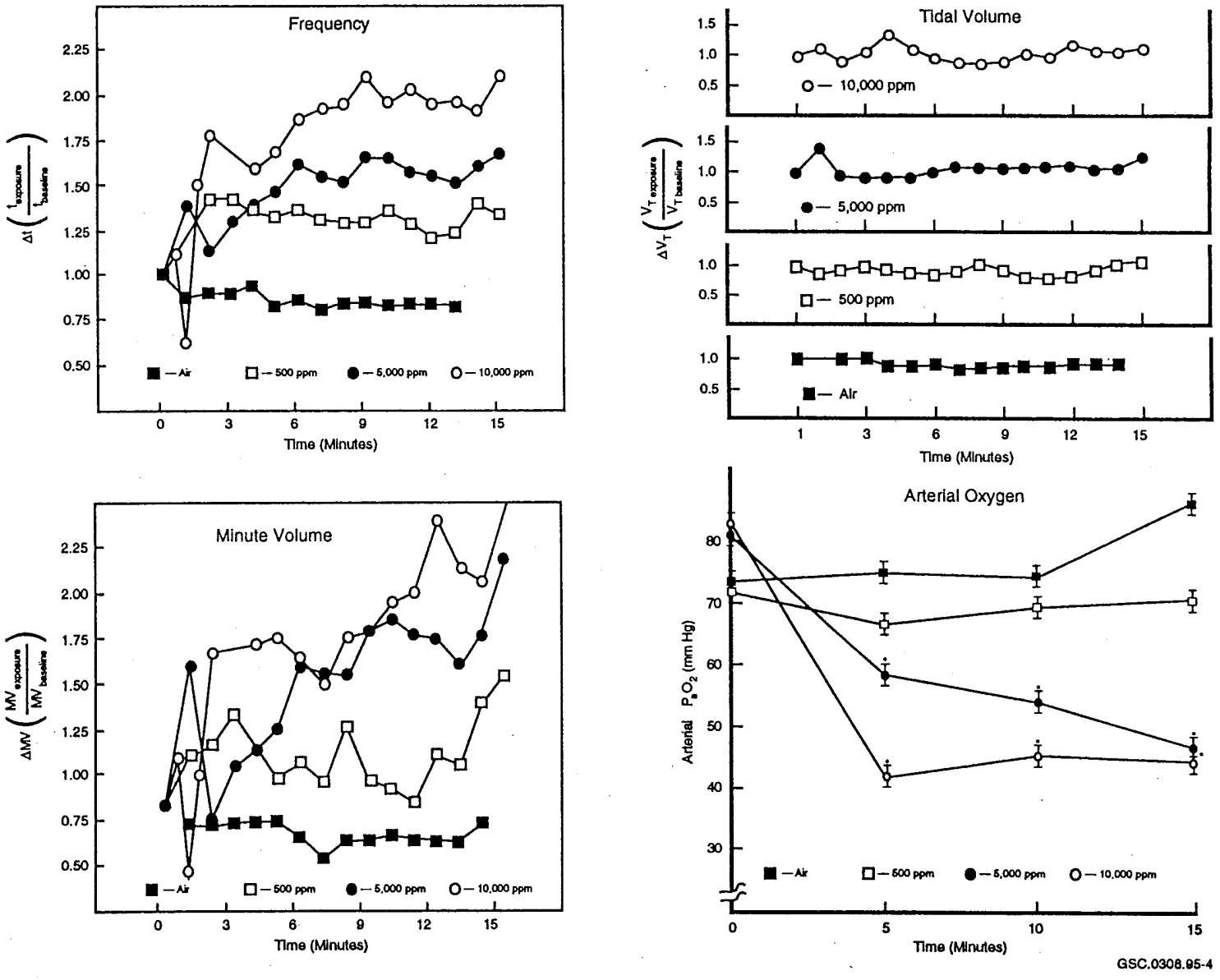
HYDROGEN CHLORIDE. Kaplan et al.[17] exposed juvenile African Savannah baboons to HCl concentrations ranging from 190 to 17,290 ppm for 5 minutes. Despite irritant effects, all animals successfully performed the escape task. Significant postexposure effects were not observed at concentrations from 190 to 11,400 ppm. However, at the two highest concentrations, 16,570 and 17,290 ppm, severe dyspnea persisted after exposure and the animals died 18 and 76 days postexposure, respectively. Thus, the LC_{50} for this method is probably somewhere between 11,400 and 16,570 ppm for a 5-minute exposure. Histopathic examination revealed pneumonia, pulmonary edema, and tracheitis with epithelial erosion. Irritant effects were observed at concentrations of 810 ppm and higher, increasing in severity from coughing and frothing at the mouth at the lower concentrations to profuse salivation, blinking/rubbing of the eyes, and shaking of the head at higher concentrations.

With a comparable shuttlebox and escape paradigm for rats, 5-minute exposures to HCl (11,800 to 76,730 ppm) did not prevent escape. However, the rat exposed to 87,660 ppm collapsed at 4 minutes 13 seconds and was dead at approximately 5 minutes of exposure. The rats exposed to 11,800 and 14,410 ppm experienced respiratory difficulties for several days. But severe postexposure respiratory effects and lethality occurred at 15,250 ppm and higher. Thus, Kaplan et al. have shown that the dose required for a 5-minute incapacitation (383,650 ppm•min to 438,300 ppm•min) greatly exceeds the minimum 5-minute dose required for subsequent respiratory failure and death (72,050 ppm•min to 76,250 ppm•min).

The results of escape performance tests with baboons are consistent with rat escape performance data and suggest that laboratory tests with rodents may be used to predict escape capability of humans exposed to HCl. Likewise minimum doses required for subsequent death are consistent for baboons and rats. Exposure to high concentrations of irritant gasses such as HCl may result in escape, only to result in a subsequent death. Minimum dose for subsequent death (for HCl) appears to be more meaningful than dose required for incapacitation (or escape) for gauging survivability in aircraft cabin fires. Yet t_i is also meaningful in that HCl can contribute to incapacitation in the aircraft.

In a later study, Kaplan et al.[18] exposed each of 4 groups of 3 anesthetized baboons in a head only mode for 15 minutes to air or 1 of 3 HCl concentrations averaging 562, 4085, and 10,170 ppm. The baboons were able to survive short exposures to high concentrations of HCl without any significant effects on pulmonary function during the 3 months after exposure. The data are illustrated in figure 14. The respiratory rate (f) increased as the HCl concentrations increased. There is no significant change in the tidal volume (V_T) with increasing concentrations. The tidal volume is the volume of air exhaled in each breath. The respiratory minute volume (rmv) increases as the concentration of HCl increases. Despite the increased respiratory frequency and minute volume, arterial P_aO_2 values decreased rapidly in the animals of the two higher HCl exposure groups. Arterial P_aO_2 appeared to return to normal levels when measured at 3 days and also at 3 months postexposure. This increase in RMV contrasts to a decrease in the RMV of rodents exposed to HCl which reduces the uptake of other gases and delays incapacitation.[10]

Hartzell et al. exposed groups of 6 to 8 rats to a range of concentrations for 5, 10, 15, 22.5, 30 and 60 minutes using a head only exposure in a National Bureau of Standards smoke toxicity test chamber modified by incorporation of a head isolation system. Postexposure lethality was monitored for 14 days[19].



Reprinted with permission from the Journal of Toxicology and Environmental Health Copyright © 1988 by Hemisphere Publishing Co., New York.[18]

FIGURE 14. EFFECT OF HYDROGEN CHLORIDE ON ANESTHETIZED BABOON RESPIRATION (KAPLAN)

Experimental $L(Ct)_{50}$ values for HCl varied from 79,500 ppm•min (5-minute exposure to 15,900 ppm) to 168,600 ppm•min (60-minute exposure to 2,810 ppm) (table 5, figure 15). Thus, Haber's Rule does not hold for HCl exposures. Plotting LC_{50} HCl vs 1/time of exposure results in a linear curve as illustrated in figure 16.

TABLE 5. SUMMARY OF HARTZELL'S HYDROGEN CHLORIDE LETHALITY DATA FOR RESTRAINED RATS

Exposure Time (min)	LC_{50} (ppm)	$L(Ct)_{50}$ (ppm•min)
5	15,900	79,500
10	8370	83,700
15	6920	103,800
22.5	5920	133,200
30	3715	111,450
60	2810	168,600

Hartzell's rat 5-minute LC_{50} (15,900 ppm) is consistent with Kaplan's rat (about 15,250 ppm) and primate (11,400-16,570 ppm) escape task LC_{50}s. Hartzell obtained a regression equation to fit his data.

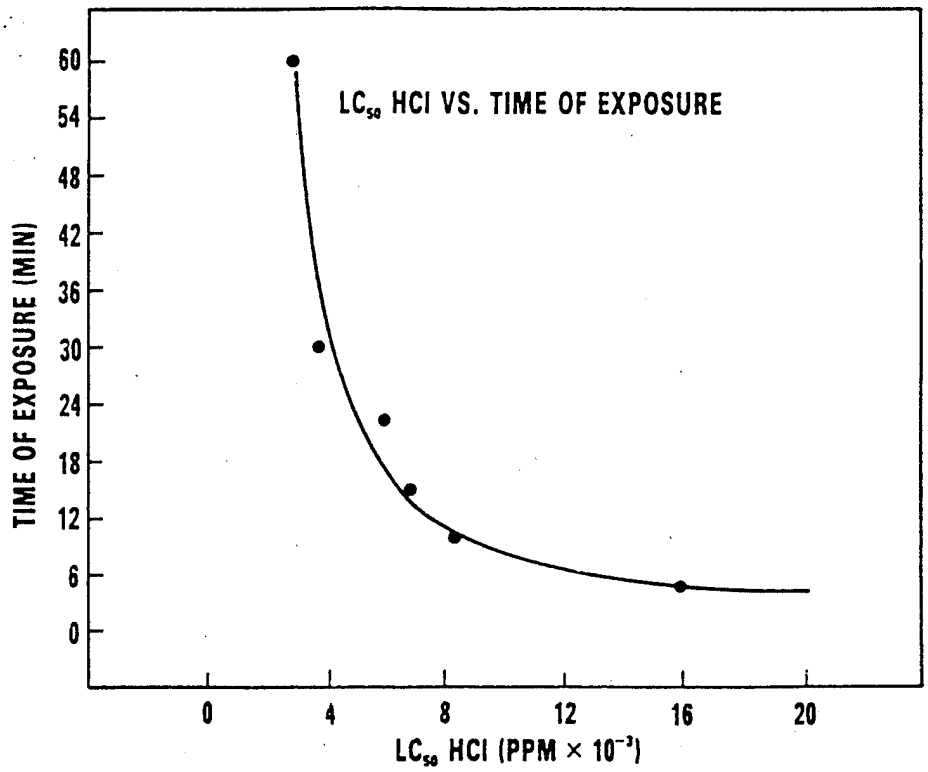
$$t_{exposure\ HCl} = \frac{70{,}500}{C_{HCl} - 1800} \quad \text{where } C_{HCl} > 1800 \text{ ppm} \quad (14)$$

(*in minutes*)

Crane et al. exposed 43 male albino Sprague-Dawley rats in a motor-driven rotating wheel to concentrations from 2000 to 100,000 ppm HCl. However, Crane exposed the rats at these concentrations for a duration required for incapacitation. For example, an exposure of 2300 ppm resulted in incapacitation at 185 minutes; an exposure of 14,700 ppm resulted in incapacitation in 32.5 minutes; and an exposure of 94,000 ppm resulted in incapacitation in 5.5 minutes. Crane obtained a regression equation to fit his data.

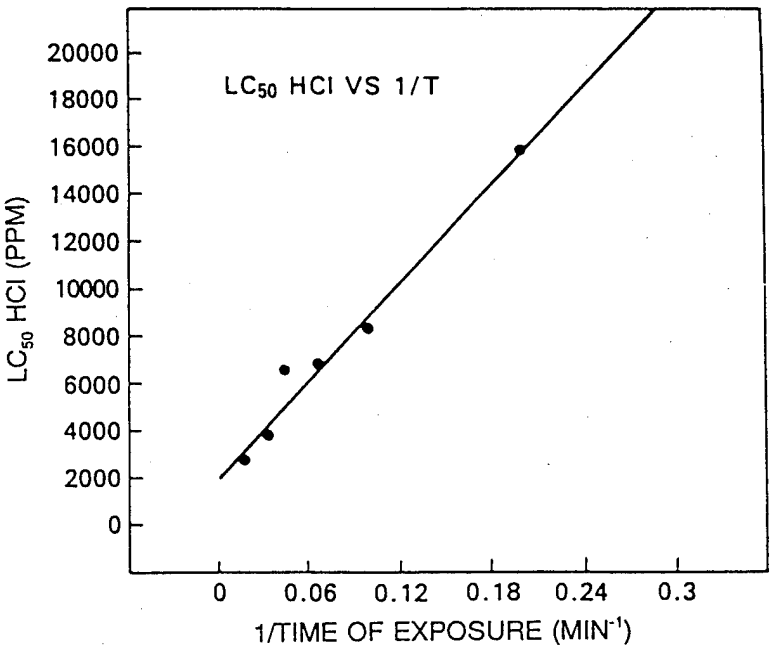
$$t_{i\ HCl} = 3 + \frac{3.36 \times 10^{+5}}{(C_{HCl} - 300)} \quad \text{when } C_{HCl} > 300 \text{ ppm}$$
$$\text{where } C_{HCl} \text{ is in ppm, and } t_i \text{ is in minutes} \quad (15)$$

This data along with the best fit curve to the data is illustrated in figures 17 and 18[20].



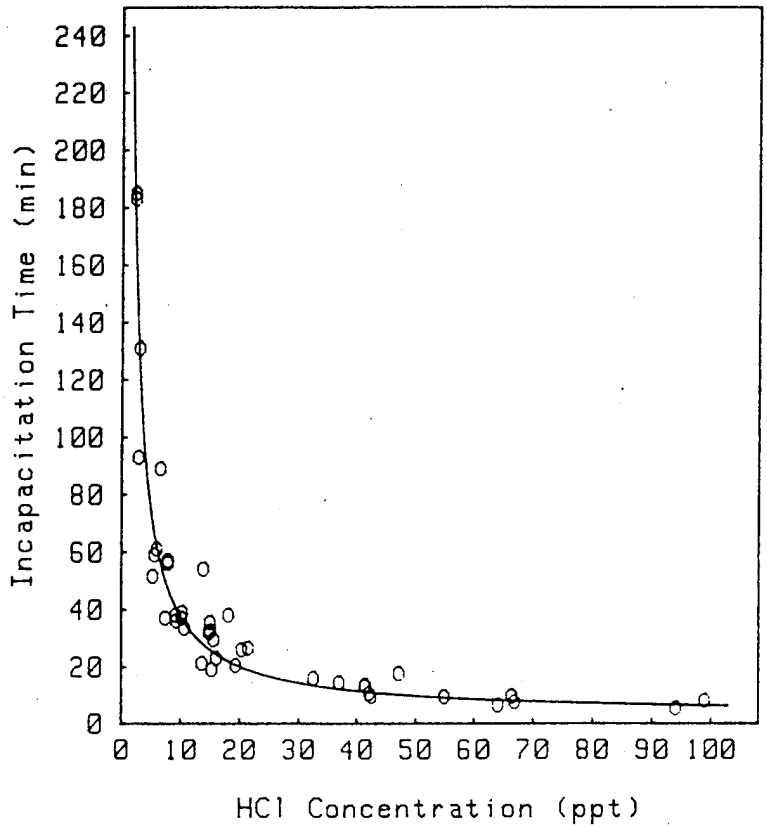
Reprinted with permission from the Journal of Fire Sciences, Copyright 1985 by Technomic Publishing Co., Inc.[19]

FIGURE 15. RELATIONSHIP BETWEEN EXPOSURE TIME AND LC_{50}s FOR RESTRAINED RATS EXPOSED TO HYDROGEN CHLORIDE (HARTZELL)



Reprinted with permission from the Journal of Fire Sciences, Copyright 1985 by Technomic Publishing Co., Inc.[19]

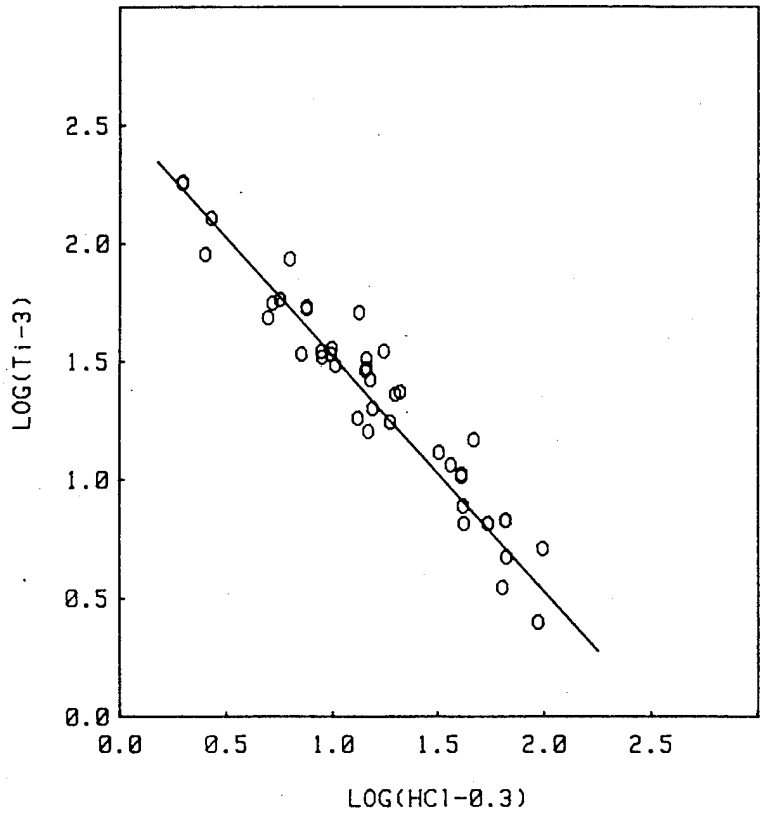
FIGURE 16. RELATIONSHIP BETWEEN $1/t_{exposure}$ AND LC_{50}s FOR RESTRAINED RATS EXPOSED TO HYDROGEN CHLORIDE (HARTZELL)



10 ppt = 10,000 ppm = 1%

Note: Concentration units are in parts per thousand

FIGURE 17. TIME TO INCAPACITATION FOR ACTIVE RATS EXPOSED TO HYDROGEN CHLORIDE (CRANE)[20]



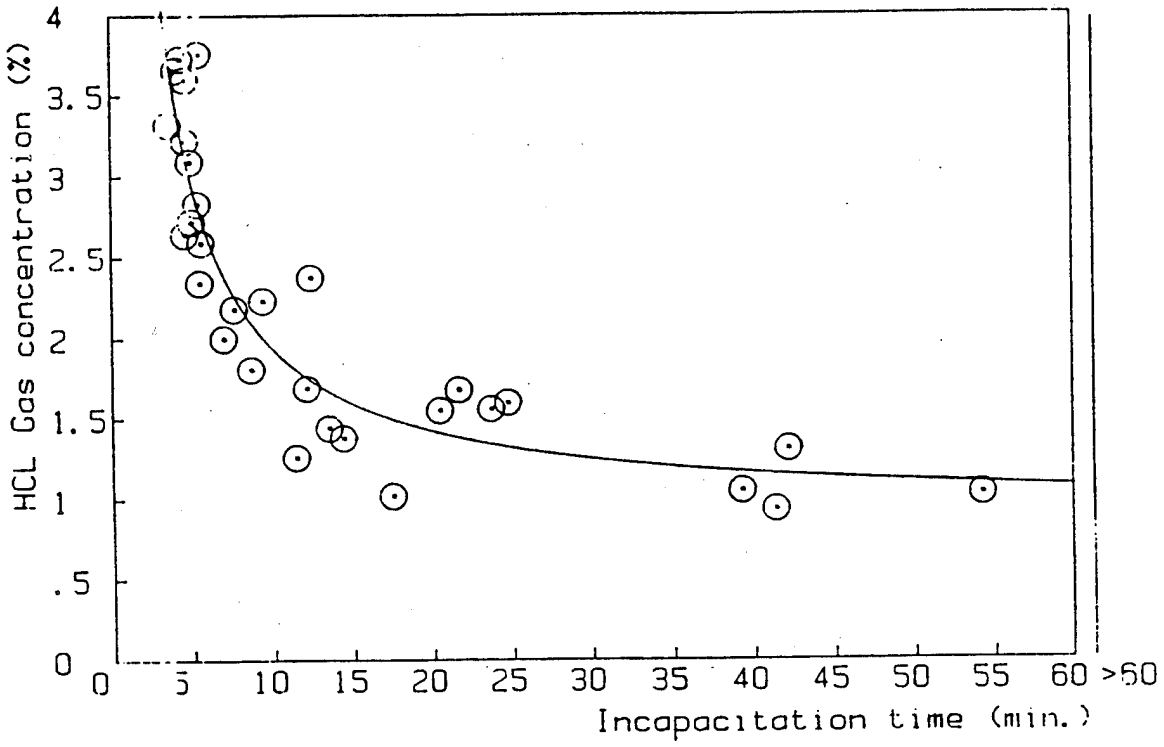
Note: Concentration units are in parts per thousand

FIGURE 18. PLOT OF LOG (t_i-3) VERSUS LOG (C_{HCl}-0.3) FOR ACTIVE RATS (CRANE)[20]

Crane et al. have shown that the HCl concentration required for incapacitation far exceeds the concentration required for subsequent respiratory failure and death for a given exposure time[20]. The mechanism of action for HCl toxicity in rats, based on observations during this study, seems to be primarily one of mechanical blockage of the upper airways (nasal pharyngeal) caused by the extreme inflammatory and corrosive action of this strong mineral acid on these tissues. Postmortem examination and gross pathology performed by a forensic pathologist indicated surprisingly little damage to airways below the trachea but almost total destruction from pharynx to nares.

Sakurai[9] exposed mice in motor-driven rotating wheels to various HCl concentrations. Eight mice were exposed for each concentration. A plot of HCl concentration versus time to incapacitation can be found in figure 19. He obtained a regression equation to fit this data.

$$t_i = \frac{1.03 \times 10^{+5}}{C_{HCl} - 9040} \qquad \text{when } C_{HCl} > 9040 \text{ ppm} \tag{16}$$



Reprinted with permission from the Journal of Fire Sciences, Copyright 1989 by Technomic Publishing Co. Inc.[9]

FIGURE 19. TIME TO INCAPACITATION FOR ACTIVE MICE EXPOSED TO HYDROGEN CHLORIDE (SAKURAI)

HYDROGEN FLUORIDE. A 1971 paper by Higgins et al.[21] presents data obtained on single gas exposures of HF and HCl to rats and mice. The LC_{50} of HF is 2.2 times less than the LC_{50} of HCl for

5-minute exposures. HF and HCl exposure concentrations were controlled by continuous monitoring using specific ion electrodes. A summary of these data can be found in table 6.

TABLE 6. SUMMARY OF HIGGINS' HYDROGEN FLUORIDE AND HYDROGEN CHLORIDE LETHALITY DATA

Gas	Animal	5-Minute LC_{50} (ppm)
HF	Rats	18,200
HCl	Rats	40,989
HF	Mice	6,247
HCl	Mice	13,745

However, HCl LC_{50}s discussed in this report are markedly higher than HCl LC_{50}s in recent literature (see previous section on HCl).

A 1976 paper by Wohlslagel et al.[22] presents data obtained on single gas exposures of HF and HCl to rats and mice. The HF LC_{50} was found to be 2.2 times lower than the HCl LC_{50} for a 60-minute exposure. Gas concentrations in the chamber were monitored during the exposures. The exposure chamber was a modified Longley type utilizing a sliding cage drawer to permit rapid insertion and withdrawal of test animals from the contaminant equilibrated chamber. A summary of this study can be found in table 7.

TABLE 7. COMPARISON OF WOHLSLAGEL'S HYDROGEN FLUORIDE AND HYDROGEN CHLORIDE LETHALITY DATA

Gas	Animal	60-Minute LC_{50} (ppm)
HF	Rats	1395
HCl	Rats	3124
HF	Mice	342
HCl	Mice	1108

Assuming $LC_{50\ HCl} = 2.2 \times LC_{50\ HF}$, the regression equation for Hartzell's LC_{50} HCl data (equation 14) can be rearranged.

$$t_{exposureHF} = \frac{32{,}045}{C_{HF} - 818.0} \quad \text{when } C_{HF} > 818 \text{ ppm} \quad (17)$$

(*in minutes*)

Data for HF incapacitation is also lacking. Assuming for a given t_i, $C_{HCl} = 2.2 \times C_{HF}$, the regression equation for Crane's incapacitation data (equation 15) can be rearranged.

$$t_{iHF} = 3.0 + \frac{1.53 \times 10^{+5}}{C_{HF} - 136.0} \quad \text{when } C_{HF} > 136 \text{ ppm} \quad (18)$$

(*in minutes*)

HYDROGEN BROMIDE. No recent data are available on HBr toxicity. However, the toxicity should be similar to that of HCl due to the chemical similarity.

ACROLEIN. Kaplan et al.[17] exposed juvenile African Savannah baboons to acrolein (CH_2CHCHO) concentrations ranging from 12 to 2780 ppm for 5 minutes apiece. These acrolein levels neither prevented escape nor affected escape times despite irritant effects at all concentrations of acrolein. Symptoms ranged from blinking and closure of the eyes and rubbing of the eyes/nose at lower concentrations to salivation, nasal discharge, violent shaking of the head, and nausea at the higher concentrations. The animal exposed to 1025 ppm died the day following exposure and the animal exposed to 2780 ppm expired approximately 1 1/2 hours after exposure. Pulmonary damage was evident in both animals but was particularly severe in the latter animal. Postexposure symptoms were evident in 1 of the 2 animals exposed to 505 ppm acrolein. The lethal 5-minute concentration for acrolein is probably somewhere between 505 and 1025 ppm. Assuming the lethal concentration to be 765 ppm, the average of these values, and assuming Haber's Rule to hold:

$$C_{Acrolein} \cdot t_{exposure\ Acrolein} = 3825\ ppm \cdot min \quad (19)$$

Skogg[23] exposed rats for 30-minute periods to acrolein concentrations of 45 to 310 ppm establishing a 30-minute LC_{50} of 135 ppm. Assuming Haber's Rule holds:

$$C_{Acrolein} \cdot t_{exposure\ Acrolein} = 4050\ ppm \cdot min \quad (20)$$

Pattle and Cullumbine[24] reported the LC_{50} for mice, guinea pigs, and rabbits to be 10.5 ppm for a 6-hour exposure. Assuming Haber's Rule holds:

$$C_{Acrolein} \cdot t_{exposure\ Acrolein} = 3780\ ppm \cdot min \quad (21)$$

Other workers have explored the biochemical effects on, and the adaptation of rats exposed to extremely low levels of acrolein[25,26]. Speitel averaged Skogg's LC_{50} of 4050 ppm and the LC_{50} of Pattle and Cullumbine of 3780 ppm:

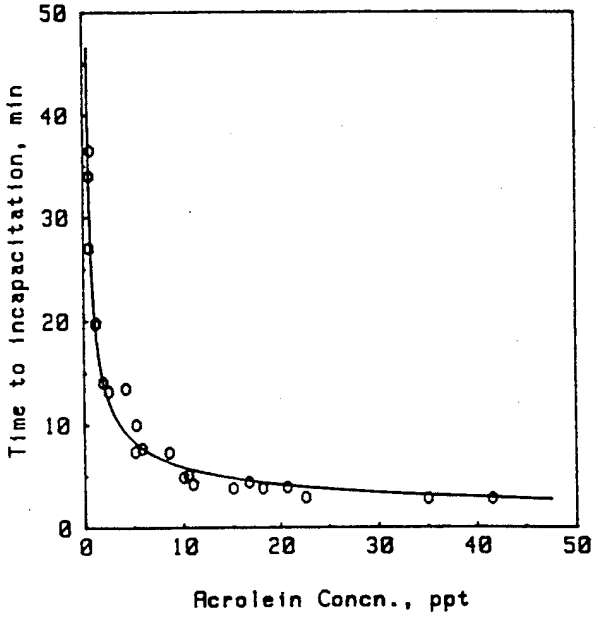
$$C_{Acrolein} \cdot t_{exposure\ Acrolein} = 3915\ ppm \cdot min \quad (22)$$

For a given exposure time, the LC_{50} is much less than the concentration required for incapacitation. Thus it is possible to survive an acrolein exposure, only to die subsequently from pulmonary damage. For this reason LC_{50} is believed to be a better indicator of ability to survive for acrolein exposures.

Crane did a study of acrolein exposures utilizing rats in a motor-driven rotating wheel to establish t_i and time to death, t_d, for concentrations ranging from 530 to over 40,000 ppm[27]. A total of 22 rats were exposed, one at a time. At concentrations around 1000 ppm, gasping began at 3 to 6 minutes, becoming severe by 6 to 11 minutes. Walking appeared normal for the first 6 to 10 minutes, after which there would be alternating periods of crawling and walking. This crawling was a sufficiently coordinated type of locomotion that it was not scored as incapacitation. The t_i's occurred in the range of 14 to 20 minutes, and severe convulsions began about 1 minute before death. This data along with the best fit curve to the data is illustrated in figures 20 and 21. An incomplete listing of test results can be found in table 8.

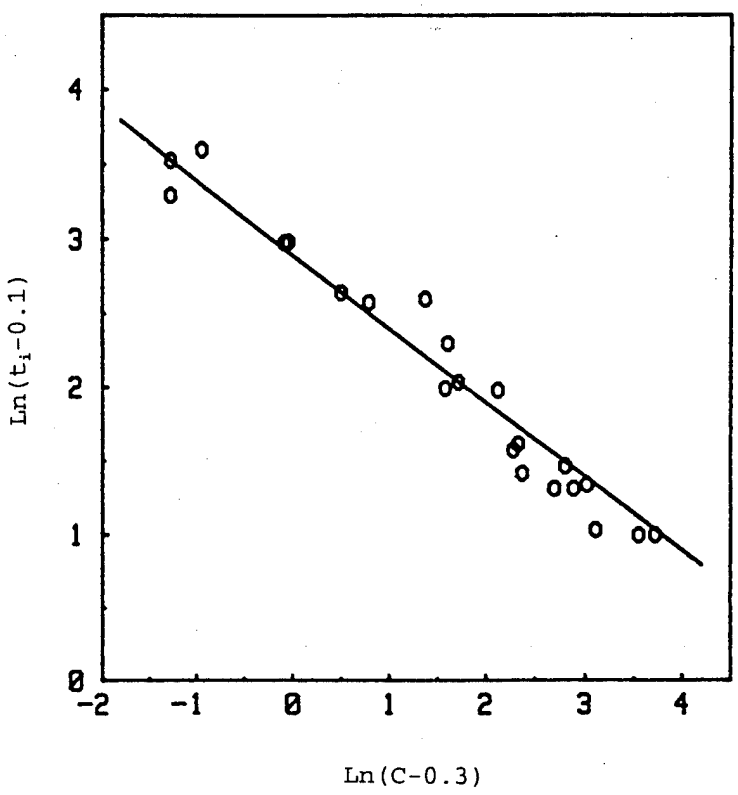
TABLE 8. SUMMARY OF CRANE'S ACROLEIN RAT DATA

Acrolein Concentration (ppm)	t_i (min)	Ct_i (ppm•min)	t_d (min)	Ct_d (ppm•min)
35,010	2.8	98,028	4.9	171,549
10,540	5.1	53,754	6.7	70,618
5280	10.0	52,800	20.2	106,656
1260	19.8	24,948	28.6	36,036
690	36.5	25,185	56.2	38,778



Note: Concentration units are in parts per thousand

FIGURE 20. TIME TO INCAPACITATION FOR ACTIVE RATS EXPOSED TO ACROLEIN (CRANE)[27]



Note: Concentration units are in parts per thousand

FIGURE 21. PLOT OF ln (t_i -0.1) VERSUS ln ($C_{Acrolein}$ -0.3) FOR ACTIVE RATS (CRANE)[27]

It is interesting to note that the C•t product decreases as the concentration increases.

The fitted regression equations for Crane's data is:

$$t_i = 1.50 + (4.0 \times 10^{+4})/(C + 500) \quad (23)$$

$$t_d = 3.75 + (5.0 \times 10^{+4})/(C + 500) \quad (24)$$

where t_i and t_d are expressed in minutes and concentration is in parts per million. Note that the time to death, t_d, is based on an exposure up to time of death.

Iwanoff[4] exposed cats to approximately 10 ppm acrolein for 3.5 hours. Symptoms included irritation, salivation, lacrimation, and mild narcosis. The animals returned to normal in a few hours.

Acrolein has been measured semiquantitatively in FAA full-scale aircraft fire tests. An estimated maximum concentration of 100 ppm was measured.

NITROGEN DIOXIDE. Kimmerle compiled data on human exposures to nitrogen dioxide. Nitrogen dioxide concentrations of 90 ppm resulted in pulmonary edema after 30-minute exposures. One hundred

to 200 ppm was found to be very dangerous within 30 to 60 minutes. Exposures to 250 ppm results in death after a few minutes.

Creasia exposed young adult female mice to 170 ppm NO_2 for 5 hours. The mice developed edema within 2 hours of the beginning of the exposure. The water content in the lungs of these animals immediately after exposure was nearly 4.5 times that in the control animals. Twelve days after the single NO_2 exposure the average water content of exposed lungs was 2.5 times that of the lungs of the control mice. These mice also developed marked focal hyperplasia (an increase in the number of cells whereby the bulk of the part of the organ is increased) in the TB/AD region. This hyperplasia progressed through day 14 after exposure. The lesion induced by a single exposure to 170 ppm NO_2 was not observed to regress for at least 70 days. At 70 days, the NO_2 induced lesion could be characterized as focal fibrotic nodules[28].

Carson et al.[29] obtained a 30-minute LC_{50} for NO_2 approximating 162 ppm for rats of 100 to 120 g.

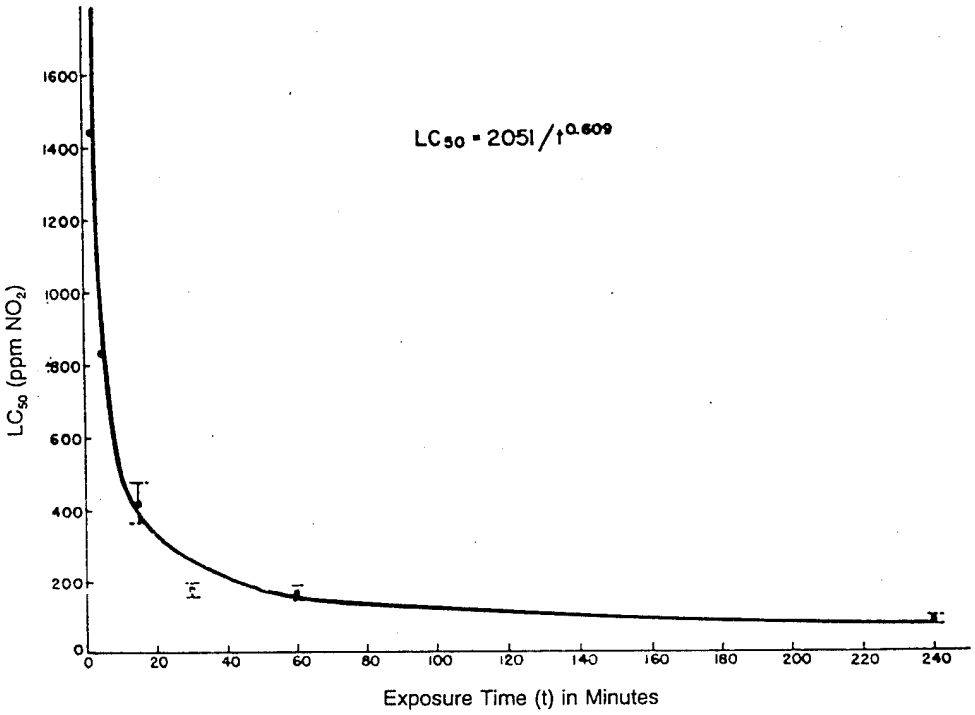
Gray et al.[30] obtained LC_{50}s for 2-, 5-, 15-, 30-, 60-, and 240-minute exposures of unrestrained albino rats weighing 200 to 300 grams. The rats were exposed in groups of 10. He plotted LC_{50}s vs. exposure time as listed in table 9 and illustrated in figure 22. Gray gave the following regression equation as the best fit to his data.

$$LC_{50} = \frac{2051}{t^{0.609}} \tag{25}$$

Higgins et al.[31] exposed 250 to 275 gram rats to a series of NO_2 concentrations (10 rats per exposure) and determined the 5-minute LC_{50} to be 831 ppm.

TABLE 9. ACUTE EXPOSURES OF UNRESTRAINED RATS TO NITROGEN DIOXIDE (GRAY ET AL.)

Number of Runs	Exposure Time (min)	LC_{50} (ppm)
3	2	1445
2	5	833
6	15	420
10	30	174
10	60	168
7	240	88



Reprinted with permission of the Helen Dwight Reid Educational Foundation, from Archives of Environmental Health, formerly known as A.M.A. Archives of Industrial Hygiene and Occupational Medicine, Copyright 1954 by Heldref Publication.[30]

FIGURE 22. RELATIONSHIP OF LC_{50}s TO EXPOSURE TIME FOR MALE RATS EXPOSED TO NITROGEN DIOXIDE (GRAY)

Levin et al. exposed 54 male Fischer 344 rats to approximately 200 ppm NO_2 for 30 minutes. The LC_{50} value for NO_2 was 200 ppm with 95% confidence limits of 190 to 210 ppm. All deaths from NO_2 exposures occurred during the postexposure period between 90 minutes and 24 hours. All animals were observed 14 days postexposure. The animals appeared normal immediately following exposure, although some occasional rapid breathing was noted. In some cases, the animals were observed to become extremely agitated just prior to death, and after death, foamy liquid was noted coming from the openings of the nostrils.[32]

Speitel derived an equation to provide the best fit for Levin's, Gray's, and Higgins' data. This best fit is illustrated in figure 23.

$$t_{exposure\ NO_2} = \frac{3899}{C_{NO_2} - 71.8} \qquad \text{where } C_{NO_2} > 71.8 \text{ ppm} \tag{26}$$

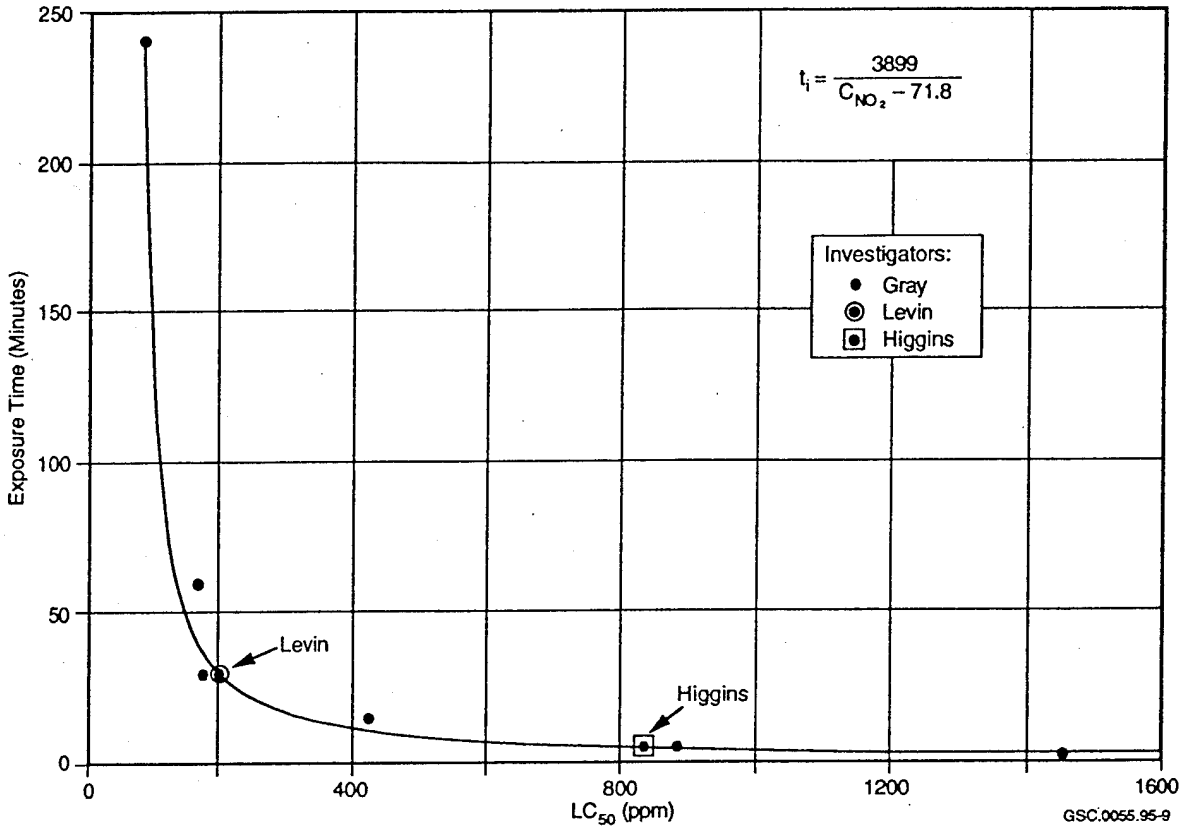


FIGURE 23. NITROGEN DIOXIDE EXPOSURES FOR RATS (GRAY, HIGGINS, LEVIN)

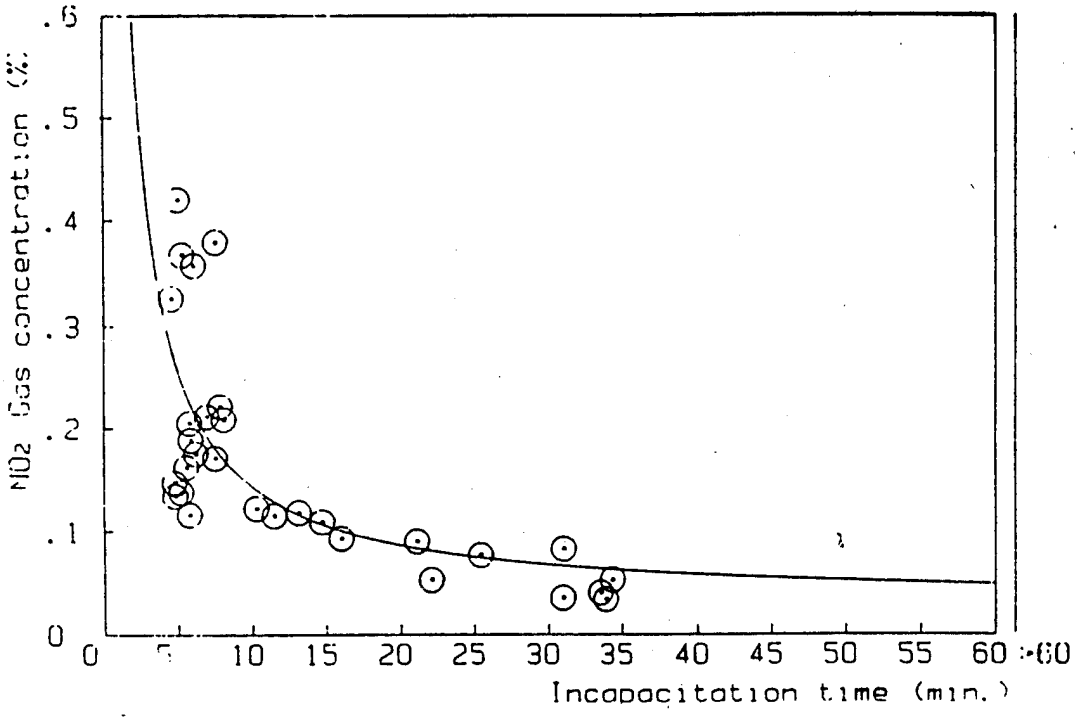
In the same study, Levin et al. determined that the LC_{50} value for NO_2 in the presence of 5% CO_2 decreased to 90 ppm with 95% confidence limits of 70 to 120 ppm. This LC_{50} was also based on 9 experiments during which the animals initially appeared unaffected except for the rapid breathing noted in most of the animals.

Sakurai[9] exposed 30 mice in motor-driven rotating wheels to NO_2 concentrations ranging from 300 to 4000 ppm. He plotted gas concentrations versus time to incapacitation as illustrated in figure 24. He derived a regression equation to fit this data.

$$t_{i\,NO_2} = \frac{1.14 \times 10^{+4}}{C_{NO_2} - 290} \qquad \text{where } C_{NO_2} > 290 \text{ ppm} \qquad (27)$$

(*in minutes*)

SULFUR DIOXIDE. Kimmerle reported human symptoms resulting from sulfur dioxide (SO_2) exposures. Concentrations of 20 ppm result in coughing and eye irritation. Concentrations from 100 to 250 ppm were found to be dangerous to life. Concentrations from 600 to 800 ppm resulted in death in a few minutes.



Reprinted with permission from the Journal of Fire Sciences, Copyright 1989 by Technomic Publishing Co., Inc.[9]

FIGURE 24. TIME TO INCAPACITATION FOR ACTIVE MICE EXPOSED TO NITROGEN DIOXIDE (SAKURAI)

Sulfur dioxide was shown to be far less toxic in the following study of mice: Bitron and Aharonson[33] studied the delayed mortality of mice following inhalation of acute doses of SO_2. One hundred eighty-two mice were exposed to a concentration of 1400 ppm SO_2 for durations ranging from 15 to 180 minutes. The animals were monitored for 45-days postexposure for delayed mortality. A best fit was performed on the final cumulated mortality as a function of exposure time. A 15-minute exposure time resulted in 15% mortality. A 30-minute exposure resulted in 40% mortality. A 38-minute exposure resulted in 50% mortality. A 60-minute exposure resulted in 70% mortality. A 150-minute exposure resulted in 95% mortality. In almost all experiments, deaths occurred either suddenly during exposure or at least by 5-days postexposure. The dose required to achieve 50% delayed mortality at 1400 ppm SO_2 is 53,200 ppm•min (38-minute exposure). Haber's Rule does not appear to apply for SO_2 in this study.

Other data collected in this study included:

900 ppm for 270 min (243,000 ppm•min) resulted in 67% mortality.
1400 ppm for 60 min (84,000 ppm•min) resulted in 72% mortality.
1900 ppm for 15 min (28,500 ppm•min) resulted in 71% mortality.

It is interesting to note that the Ct product decreases as the concentration increases. Speitel derived a regression equation to fit this data.

$$t_{70DD} = exp\ [8.18012 - (2.89037\ x\ 10^{-3})\ C_{SO_2}] \quad \text{where } C_{SO_2} > 300 \text{ ppm} \quad (28)$$
(in minutes)

The exposure time resulting in 70% mortality (including delayed deaths) is denoted by t_{70DD}. Since a 38-minute exposure of 1400 ppm SO_2 produced 50% mortality and a 60-minute exposure produced 70% mortality, the exposure time needed to produce 50% mortality is:

$$t_{50DD} = 0.633\ exp\ [8.18012 - (2.89037\ x\ 10^{-3})\ C_{SO_2}] \quad \text{where } C_{SO_2} > 300 \text{ ppm} \quad (29)$$
(in minutes)

CARBON DIOXIDE. At concentrations of approximately 5% and above, carbon dioxide is a narcotic. For elevated CO_2 concentrations (hypercapnia) the depth and frequency of respiration increases. From approximately 3% up to 6% there is a gradually increasing degree of respiratory distress with increasingly rapid breathing. This becomes severe at approximately 5% to 6% with clinical comments from subjects such as "breathing fails to satisfy intense longing for air;" "much discomfort, severe symptoms impending" and headache and vomiting also occurred[34]. These symptoms tend to worsen during continued exposure. Once the concentration of carbon dioxide is in the 7% to 10% plus range, a new set of symptoms consisting of dizziness, drowsiness, and unconsciousness is superimposed on the severe respiratory effects[16,35,36]. Loss of consciousness is likely within 2 minutes at 10% CO_2 in humans[1].

As with HCN and low-oxygen hypoxia, intoxication by carbon dioxide does not follow Haber's Rule. Figure 25 shows the general symptoms common to most human subjects when exposed to the times indicated to mixtures of carbon dioxide in air at a total pressure of 1 atmosphere. This chart was drawn by NASA scientists[37] from data of King[34], Nevison[38], and Schaefer[39]. In Zone 1, no psychophysiological performance degradation was noted. In Zone II, small threshold hearing losses have been found and there was a perceptible doubling in depth of respiration. In Zone III, the zone of distracting discomfort, the symptoms were mental depression, headache, dizziness, nausea, "air hunger", and a decrease in visual discrimination. Zone IV represents marked deterioration that led to dizziness and stupor, with inability to take steps for self preservation, and the final state was unconsciousness. Selected points along the Zone III, Zone IV boundary are shown in table 10.

The bar graph on the right of figure 25 shows that for prolonged exposures of 40 days, concentrations of CO_2 in air of less than 0.5% (Zone A) cause no biochemical or other effect; concentrations between 0.5% and 3% (Zone B) cause adaptive biochemical changes, which may be considered a mild physiological strain; and concentrations above 3.0% (Zone C) cause pathological changes in basic physiological functions.

Haber's Rule clearly does not apply for CO_2. Speitel derived the following expression for t_i to fit the curve defined by the Zone III, Zone IV boundary as defined by selected points in table 10.

$$t_{iCO_2} = exp\ [11.4 - 1.14\ x\ \%\ CO_2] \quad (30)$$

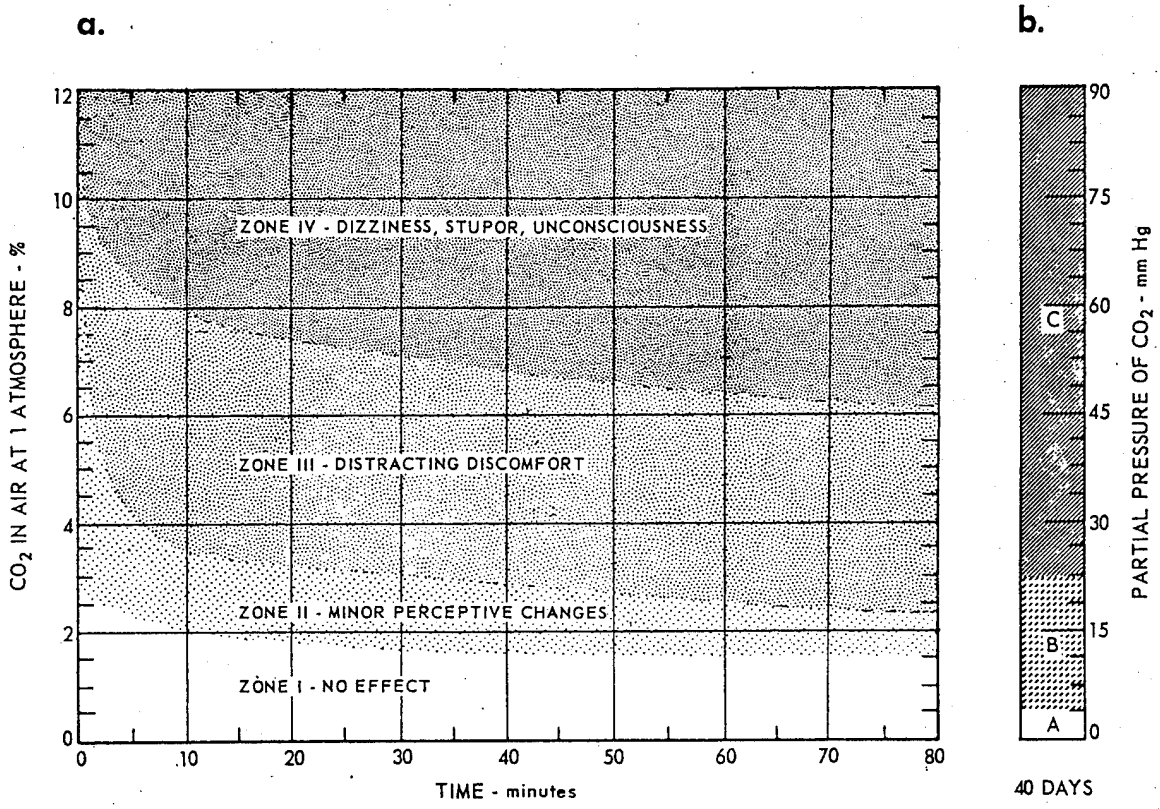


FIGURE 25. SYMPTOMS OF CARBON DIOXIDE EXPOSURE ON HUMAN SUBJECTS[37]

TABLE 10. SUMMARY OF HUMAN CARBON DIOXIDE INCAPACITATION LEVELS

Time (minutes)	Concentration CO_2 (percent)	Ct (percent minutes)
1.0	10.0	10.0
5.0	8.8	44.0
10.0	7.9	79.0
30.0	7.1	213.0
60.0	6.4	384.0
80.0	6.2	496.0

This fit is illustrated in figure 26.

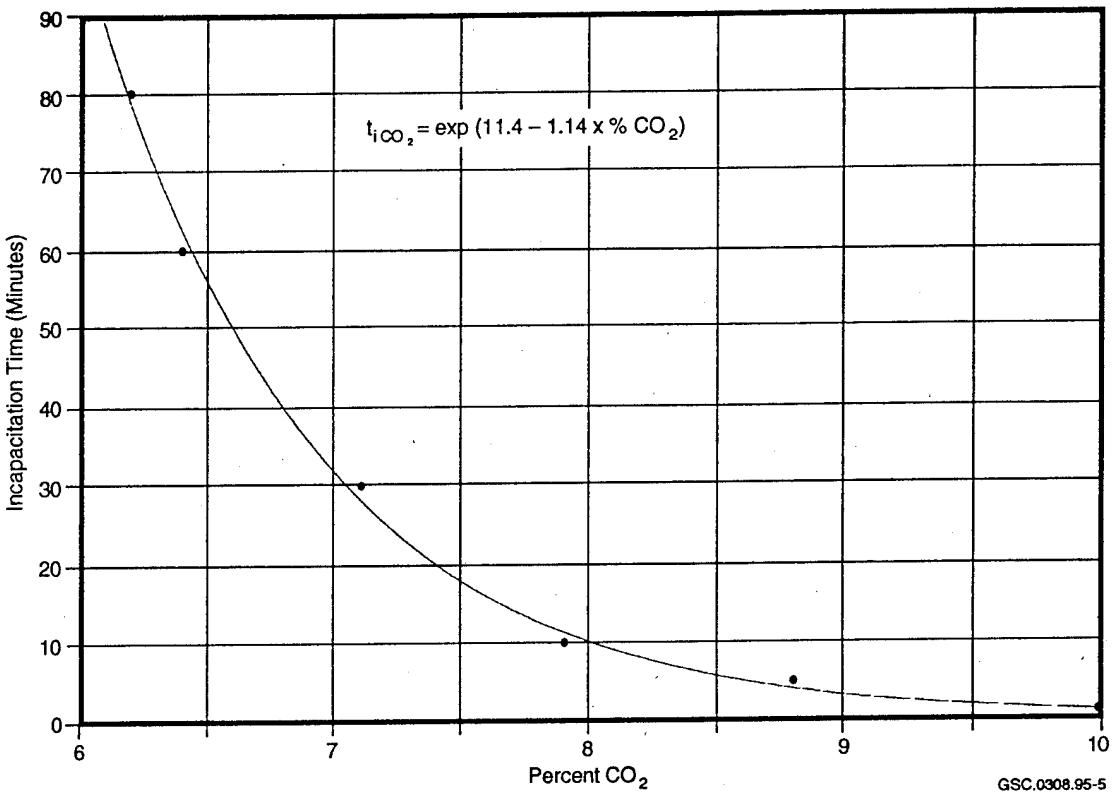


FIGURE 26. HUMAN EXPOSURES TO CARBON DIOXIDE (BLOCKLEY)

Purser[1] compiled data from Lambertson[40], Comroe et al.[41], Altman and Ditter[42], and King[34]. He derived the following expression for t_{iCO_2}.

$$t_{iCO_2} = exp\ (6.1623 - 0.5189\ x\ \%\ CO_2) \qquad (31)$$

King's references, however, indicate that the exposure times predicted by Purser's equation for 5% and 6.5% exposures do not represent the limit of endurance. For example, one of King's sources, Brown, exposed 3 human subjects to 6.0% CO_2 for 20 1/2 to 22 minutes. Brown stated the limit of endurance was not reached and the exposures could have been extended[43]. Davies, Haldane, and Kennaway[44] performed 3 separate 2-hour exposures to CO_2 concentrations: (1) 5.2%-5.6% CO_2, (2) 6.0%-6.5% CO_2, and (3) 6.0%-6.5% CO_2. The symptoms during exposure were a slight headache for the 1st experiment and none for the next two experiments. However, a headache of moderate severity was observed immediately after emerging from the chamber. In another study, Brown[45] exposed 11 human subjects to 5.3%-5.6% CO_2 for 8 hours. The effects were "severe", with labored respiration, fatigue, chilliness, and headache. Two subjects terminated exposure in the 7th hour.

A more detailed review by White[46] appeared in 1948. White's summary of CO_2 data is listed in table 11.

TABLE 11. SUMMARY DATA RELEVANT TO TOLERANCE TO CARBON DIOXIDE (WHITE)
(This data is plotted in Figure 27)

Composition of Gas Inhaled (Percent)		Summary
CO_2	O_2	
100 64.6 39.2	0 8.6 9.1	Entirely unable to inhale. (Hill & Flack - 1908)[47]
20.0	---	Immediate partial spasm of glottis with peculiar whooping sound on inspiration. (Hill & Flack - 1908)
18.6	In Air	Unconsciousness imminent in 94 and 110 seconds in 2 subjects. Extreme discomfort, mental dullness, cyanosis, throbbing in the head. (Haldane & Smith - 1892)[48]
15.3	14.5	Could inhale without spasm of the glottis for few seconds. Extremely severe dyspnea. (Hill & Flack - 1908)
12.4	39.7	Seven male subjects, aged 19 to 30 yrs., breathed mixture 0.75 to 2.0 minutes. One subject collapsed. Severe dyspnea, dizziness, and drowsiness tending to actual stupor. Choking sensation and sense of impending collapse. Dazed condition after exposure for a few minutes. (Brown - 1930)[43]
11.5	---	First inhalation unpleasant. Rapid development of dimness of vision. (Speck - 1892) (From Hill & Flack - 1908)
10.4	14.4	Seven subjects, tolerance 0.5 to 2.25 minutes. (Brown - 1930)[43]
10.4	89.6	Thirty-one subjects, 21-26 yrs. of age. Inhaled gas for an average of 3.8 minutes. Range 2.5 to 6.0 minutes, 3 unconscious, 1 analgesia, and 2 similar to onset of NO_2 anaesthesia. Two completely unaware of surroundings, 7 felt about to faint, and 18 experienced dizziness. Dyspnea (M.R.V. 40 to 140 liters per minute), restlessness, mental clouding, dimness of vision, muscle twitching, tingling, and exhaustion, mental depression, and substernal pain. Subjects stuporous in a dazed condition sitting on a stool or bicycle. Recovery rapid in 1 to 2 minutes. (Personal Communication) (Dripps and Comroe - 1941)
8.8	38.1	Four subjects. Inhaled gas 7 to 10 minutes. Very near tolerance limit. (Brown - 1930)[43]

TABLE 11. SUMMARY DATA RELEVANT TO TOLERANCE TO CARBON DIOXIDE (WHITE) (Continued)
(This data is plotted in Figure 27)

Composition of Gas Inhaled (Percent)		Summary
CO_2	O_2	
8.5	--	Opinion after reviewing literature - insupportable after 15 to 20 minutes. (Fullman - 1926)
7.6	92.4	Forty-two subjects, 21 to 26 yrs of age, inhaled gas for an average of 7.4 minutes, range 2.5 to 10 minutes, 5 unconscious, 13 dyspneic (M.R.V. 24 to 107 liters per minute). Fourteen dizzy, 9 during inhalation. Very near tolerance. Stuporous. (Dripps & Comroe - 1947)[49]
7.5	16.0	Six subjects inhaled gas 3.5 to 6.0 minutes. Symptoms "urgent" but not limit of tolerance. (Brown - 1930)[43]
7.0	---	CO_2 inhaled air = CO_2 exhaled air. (Speck - 1892)
6.0	21.1	Five subjects inhaled gas 20.5 to 22.0 minutes. Much discomfort. Urgent panting. Not intolerable, but severe symptoms impending. (Brown - 1930)[43]
6.0	---	One subject, 3 experiments. Entered chamber with CO_2 concentration at 6.0%, CO_2 varied between 5.2% and 6.5%. Exposed 2 hours doing light manual activity. Two subjects - acute exposure - 15 minutes. No difficulty. (Davies, Haldane and Kennaway - 1920)[44]
6.2 to 5.2	---	Three subjects, 33-hour exposure. Distressing. Able to do manual and mental work. (Case and Haldane - 1941)[50]
5.3 to 5.6	14.2 to 17.8	Eleven subjects exposed 8 hours in closed space, effects "severe". Decidedly labored respiration, fatigue, chilliness, and subjective depression headache. Two subjects terminated exposure in 7th hour. Two observers exposed acutely to 5.5% to 5.8% CO_2 unable to make observations due to severe dyspnea. (Brown - 1930)[45]
6.7 to 5.2	19.2 to 20.5	Four to 77 male subjects breathed recirculated air 35 to 72 hours in sealed space without serious impairment of physical condition and efficiency. Three subjects terminated exposure. In 130 man exposures, CO_2 concentration slowly rising to 5% to 6%. Held at this level 5 to 40 hrs. CO_2 above 5% not well tolerated. Limiting value for healthy young men if exposure prolonged. Headache, nasal congestion, nausea, dry throat, hyperpnea. Sharp increase in symptoms with CO_2 above 5%. Excellent physiological, biochemical, and psychological data. (Consolazio, Fisher, Pace, Pecora, Pitts and Behnke - 1947)[51]

NOTE: Data selected to show result of acute exposure where possible.

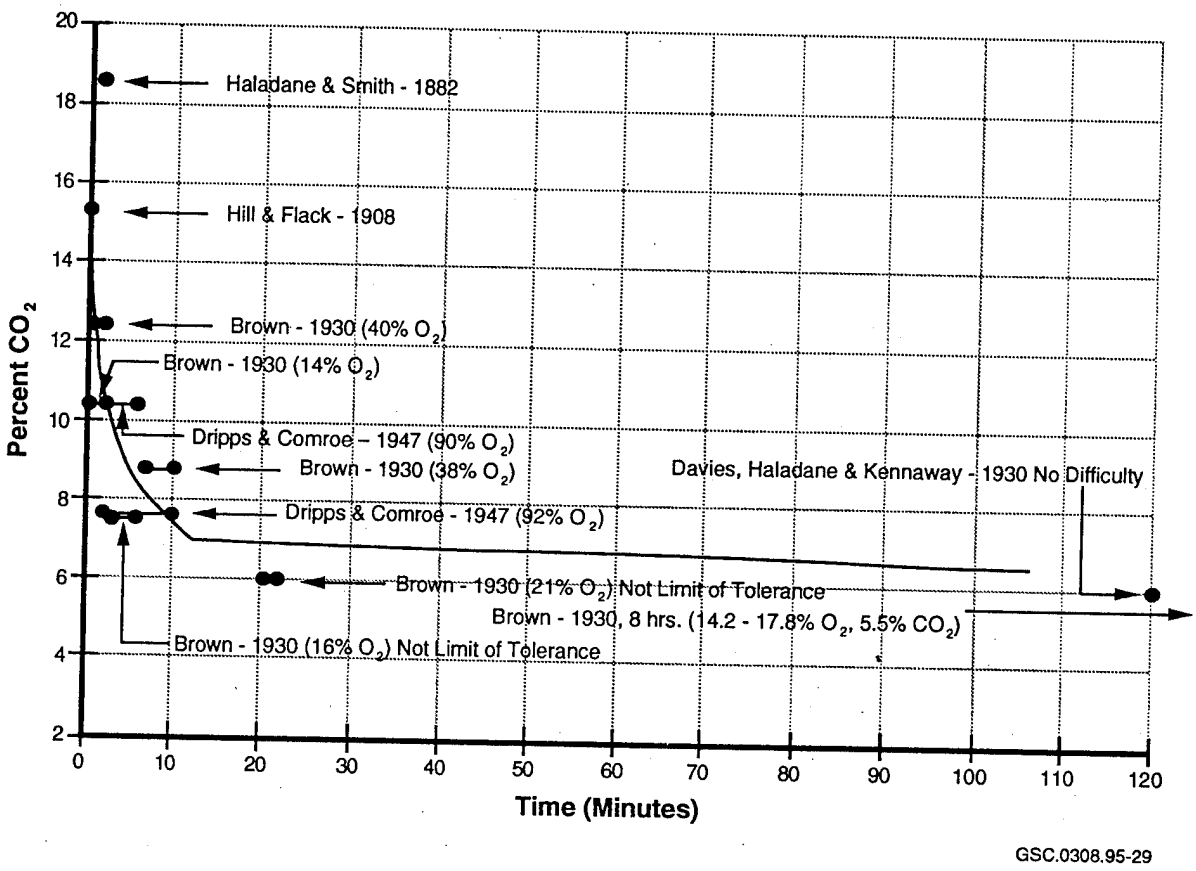


FIGURE 27. CARBON DIOXIDE GAS CONCENTRATIONS VERSUS INCAPACITATION TIME FOR HUMAN SUBJECTS

Speitel fit the lower CO_2 concentration data in figure 27 with the following equation.

$$t_i = 2193.8 - 311.6 \; x \; C_{CO_2} \qquad \text{when } 5.5 \leq C_{CO_2} \leq 7.0\% \tag{32}$$

Purser's equation (31) provides a good fit for the higher concentration data, and Speitel's linear fit is a reasonable fit for lower concentrations of CO_2 as illustrated in figure 27.

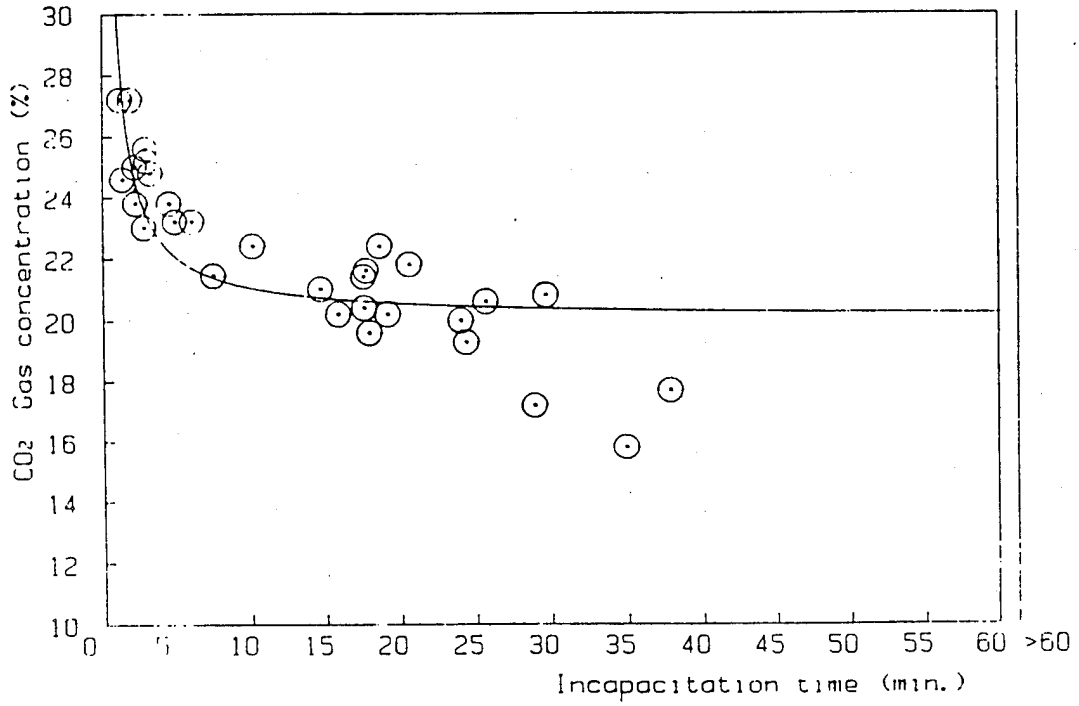
Purser exposed 4 monkeys to 5% CO_2 for 30 minutes. The monkeys sat in a chair with the test atmosphere supplied to their snouts with a face mask. No incapacitation occurred. There was approximately a three-fold increase in RMV resulting from an immediate increase in both respiratory rate and tidal volume[12].

Rats, however, are tolerant to fairly high CO_2 levels. Levin et al. subjected Fisher 344 male rats to CO_2 concentrations ranging from 1.3% to 14.7% for 30 minutes. The rats were neither incapacitated nor fatally injured. Incapacitation was indicated by lack of a righting reflex. Even rats exposed to the highest CO_2 had normal righting reflexes, posture, and exploratory behavior when tested immediately following

the exposures. In all these experiments, the O_2 levels were greater than 17% and the background CO was negligible[52]. These results agree with those of Herpol et al. who also found that rats exposed to CO_2 concentrations of up to 15% for 30 minutes never lost consciousness[53]. This indicates that far higher CO_2 concentrations are needed to incapacitate rats than humans.

Likewise, mice are also tolerant to high CO_2 levels. Sakurai et al. exposed active mice, in motor-driven rotating wheels, to CO_2 concentrations ranging from 16% to 27%. The O_2 concentration was maintained at approximately 20%. This data is plotted in figure 28. Sakurai derived the following expression for t_i.

$$t_{i\,CO_2} = \frac{9.97}{C_{CO_2} - 20.0} \qquad \text{when } C_{CO_2} > 20\% \tag{33}$$



Reprinted with permission from the Journal of Fire Sciences, Copyright 1989 by Technomic Publishing Co., Inc.[9]

FIGURE 28. TIME TO INCAPACITATION FOR ACTIVE MICE EXPOSED TO CARBON DIOXIDE (SAKURAI)

Levin et al. obtained an LC_{50} value for CO_2 of 47% for seven 30-minute exposures of 6 rats per exposure. All deaths occurred either during the 30-minute exposure period or within the following 2 minutes. The highest concentration tested at which no deaths occurred was 260,000 ppm[32]. It is clear from this data that for a given exposure time, far higher concentrations are needed to kill than to incapacitate rats.

FORMALDEHYDE. Bitron and Aharonson[33] studied the delayed mortality of mice following inhalation of acute doses of CH_2O. Two hundred and fifty-two mice were exposed to a concentration of 320 ppm CH_2O for durations ranging from 40 to 370 minutes. The animals were monitored for a 45-day postexposure period for delayed mortality. A best fit was performed on the final cumulative mortality as a function of exposure time. A 50-minute exposure time resulted in 6% mortality. A 100-minute exposure time resulted in a 50% mortality. A 150-minute exposure resulted in an 82% mortality. All deaths occurred postexposure. Not a single death was observed before the second day. Final cumulative mortalities were reached within two weeks after exposure.

The dose required to achieve 50% delayed mortality at 320 ppm CH_2O is 32,000 ppm•min.

Assuming Haber's Rule to hold for formaldehyde:

$$L(Ct)_{50\ HCHO} = C_{HCHO} \cdot t_{exposure} = K = 32{,}000\ ppm \cdot \text{min} \tag{34}$$

LOW-OXYGEN HYPOXIA. Incapacitation due to lack of oxygen, consisting of loss of consciousness, occurs when the oxygen supply to cerebral tissue falls below a certain critical value which occurs when the partial pressure of oxygen in the cerebral venous blood falls below 20 mm Hg.[54]

Shukitt et al.[55] confined thirteen male sailors and tested them in a hypobaric chamber. The sailors were exposed to normobaric oxygen concentrations of 21%, 17%, and 13% for three days at each concentration with 0.9% carbon dioxide and the balance nitrogen. It appears that normobaric oxygen concentrations as low as 17% are not likely to produce adverse effects on cognition, mood states, or acute mountain sickness (AMS). Oxygen concentrations as low as 13% are likely to adversely affect some performance tasks and moods and will induce AMS in about one-third of the exposed individuals. These effects are similar to those observed in mountain climbers at the same P_{O_2} [55].

Knight et al.[56] exposed men to 21% and 11.5% oxygen at normal atmosphere pressure. Men were also exposed to 11.5% oxygen in hyperbaric air (P_B = 1141 torr) (P_{O_2} = 131 torr). The exposure duration was 1.7 hours. Seven subjects ranging in age from 20 to 57 years participated. No deterioration in visual or mental performance was observed.

Luria and Knight[57] tested night vision (scotopic) sensitivity of six subjects while they were breathing either air (21% oxygen, P_{O_2} 158 torr) or 10% oxygen, balance nitrogen (P_{O_2} 76 torr). These tests were conducted at 1 atmosphere pressure. Continuous monitoring showed that the mean oxygen content in the arterial blood (S_aO_2) dropped from 97% to 77% during the first 7 minutes of breathing 10% oxygen and there was a significant degradation of scotopic sensitivity. Measurement started 8 minutes into the low-oxygen exposure. Each subject breathed the gas mixtures for no more than a 1/4 hour. Five subjects reported dizziness, lightheadedness, air hunger, and restlessness while breathing the 10% oxygen. (The sixth subject reported feeling lightheaded during exposure to 21% oxygen.)

Slobodnik et al.[58] exposed 12 navy divers from 25 to 48 years of age to normobaric concentrations of 6.2% O_2, 7.0% O_2, and 7.85% O_2 and the balance being N_2 respectively. A two-dimensional tracking task was used to test cognitive function of each subject while breathing a test mixture. The performance of

the tracking task fell from 100% to 70% for 25% of the subjects exposed to 6.2% O_2 at 3.5 minutes; 7.00% O_2 at 5.4 minutes; and 7.85% O_2 at 9.75 minutes. There was a wide variation of cognitive function as a function of time among the test subjects. The times for the first 25% of subjects to reach 70% of control performance at 6.2% O_2 ranged from 2.25 to 3.5 minutes. At 7.0% O_2, the times for the first 25% of subjects to reach 70% of control performance ranged from 4.0 to 5.4 minutes. Six subjects were still performing above 70% of the average control performance 9.75 minutes into the test. At 7.85% O_2, times ranged from 5.75 to 9.75 minutes for the first 25% of the subjects to fall to the 70% performance level.

Purser exposed 4 monkeys to 10% and 15% O_2 for 30 minutes. Each animal was placed in a chair and the test atmosphere was supplied to the animal's snout via a face mask. Animals exposed to 10% O_2 appeared to be superficially normal and were definitely conscious. However, there was clear evidence of muscle weakness and impairment of reflexes. Three out of the 4 animals tested suffered electrocardiogram abnormalities during exposure consisting of paroxysmal premature ventricular extrasystoles. Arterial P_{O_2} fell by 64%. Venous P_{O_2} fell to as little as 10 mm Hg. In contrast to the above, exposure to 15% oxygen appeared to have no effect on the animals beyond a 39% reduction in the arterial P_{O_2} and a slight increase in heart rate[12]. Physical exertion would be expected to greatly increase the severity of all these symptoms and signs[59].

Most low oxygen hypoxia data in the literature was taken at altitude or at hypobaric pressure. It can be argued that this data may not be valid for sea level low oxygen exposures. It can be seen from the altitude pressure table (table 12)[60] that the partial pressure of oxygen after passing through the trachea at a given altitude can be markedly different than the surroundings. Inspired gases pick up water from wet respiratory passages until the partial pressure of water vapor reaches the saturation pressure of 47 mm Hg at body temperature (98.6°F or 37°C). Assuming the inspired air has been saturated with water vapor at 37°C, the total pressure of the dry gasses is reduced to the barometric pressure B minus 47 mm Hg. It can be argued that this effect may indicate that men may be tolerant to lower level ambient concentrations at sea level than at altitude. This effect increases with increasing altitude.

In addition to the partial pressure of water, the partial pressure of CO_2 should result in a further reduction of P_{O_2} deeper into the respiratory tract in the alveoli as illustrated in figure 29[37]. As inspired gases pass into the lungs, they mix with residual air in the alveoli, lose oxygen to the blood, and pick up carbon dioxide released by the blood. The carbon dioxide mixes with the alveolar gases to an equilibrium partial pressure of 40 mm Hg. The total partial pressure of oxygen and nitrogen in the lungs is therefore 40 mm Hg less than the tracheal gas (curve B). In most subjects, the body compensates automatically (within a limited range) for low oxygen pressure by increasing the breathing rate and/or depth until the point where hypocapnia (too low of a carbon dioxide concentration) sets in. This increases slightly the partial pressure of oxygen (P_{O_2}) within the compensatory range as shown on curve D. It can be seen from this figure that at altitudes above 50,000 feet the alveoli contain only water and carbon dioxide. At an altitude of 63,000 feet, where the total pressure is 47mm Hg, the alveoli contain only water vapor.

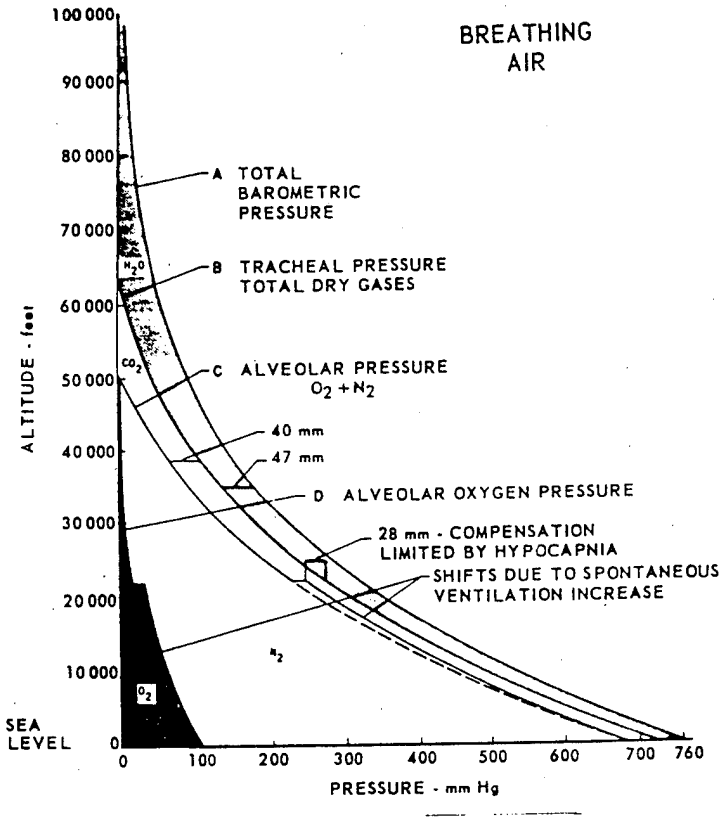
TABLE 12. ALTITUDE-PRESSURE TABLE

I Altitude, m	II Altitude, feet	III Pressure, psi	IV Pressure, mm Hg	V (B-47),* mm Hg	VI (B-47).209,† mm Hg
0	0	14.7	760	713	149
610	2000	13.7	707	660	138
1220	4000	12.7	656	609	127
1830	6000	11.8	609	562	118
2440	8000	10.9	564	517	108
3050	10,000	10.1	523	476	100
3660	12,000	9.3	483	436	91
4270	14,000	8.6	446	399	83
4880	16,000	8.0	412	365	76
5490	18,000	7.3	379	332	69
6100	20,000	6.8	349	302	63
6710	22,000	6.2	321	274	57
7320	24,000	5.7	294	247	52
7930	26,000	5.2	270	223	47
8540	28,000	4.8	247	200	42
9150	30,000	4.4	226	179	37
9760	32,000	4.0	206	159	33
10,370	34,000	3.6	187	140	29
10,980	36,000	3.3	170	123	26
11,590	38,000	3.0	155	108	23
12,200	40,000	2.7	141	94	20
12,810	42,000	2.5	128	81	17
13,420	44,000	2.2	116	69	14
14,030	46,000	2.0	106	59	12
14,640	48,000	1.8	96	49	10
15,250	50,000	1.7	87	40	8
19,215	63,000	0.9	47	0	0

From U.S. Standard Atmosphere, 1962.[60]

* (B-47) = (a) total pressure of the dry gases after the inspired gas has been saturated with water vapor at 37°C; (b) also equal to the inspired O_2 tension, $P_{I_{O_2}}$, when pure oxygen is inspired.

† (B-47).209 = inspired O_2 tension, $P_{I_{O_2}}$, when air is breathed.



Altitude (feet)	Total Pressure (mm Hg)	N_2 Percentage	H_2O Percentage	CO_2 Percentage	O_2 Percentage	O_2 Pressure (mm Hg)
AMBIENT DRY ATMOSPHERE						
0	760.0	79.02	0	0.03	20.95	159.2
5000	632.3	79.02	0	0.03	20.95	132.5
10000	522.6	79.02	0	0.03	20.95	109.5
15000	428.8	79.02	0	0.03	20.95	89.8
20000	349.1	79.02	0	0.03	20.95	73.1
22000	320.8	79.02	0	0.03	20.95	67.2
TRACHEAL GAS						
0	760.0	74.13	6.18	0.03	19.66	149.4
5000	632.3	73.14	7.43	0.03	19.39	122.6
10000	522.6	71.91	8.99	0.03	19.06	99.6
15000	428.8	70.31	10.96	0.03	18.66	80.0
20000	349.1	68.38	13.46	0.03	18.13	63.3
22000	320.8	67.46	14.65	0.03	17.85	57.4
ALVEOLAR GAS						
0	760.0	75.00	6.18	5.26	13.55	103
5000	632.3	74.06	7.43	6.01	12.49	79
10000	522.6	72.45	8.99	6.89	11.67	61
15000	428.8	70.62	10.96	7.70	10.73	46
20000	349.1	68.49	13.46	8.59	9.45	33
22000	320.8	67.27	14.65	8.73	9.35	30

FIGURE 29. PARTIAL PRESSURES OF WATER, CARBON DIOXIDE, AND OXYGEN IN AIR AT VARIOUS ALTITUDES[37]

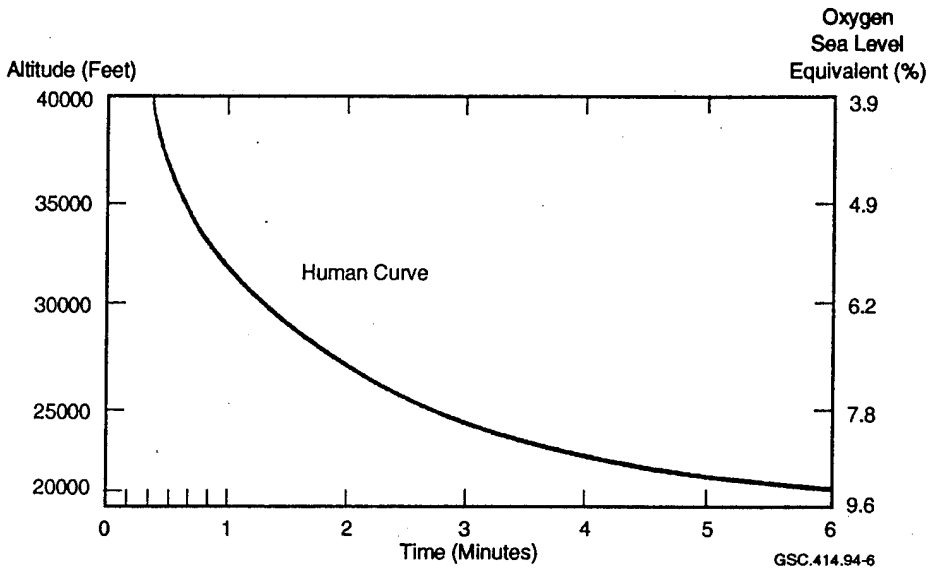
Purser adapted data from Luft[54] and plotted time of useful consciousness for men at rest following sudden decompression (< 1 second transition time) to a range of simulated altitudes, assuming similar effects to those of reduced oxygen concentrations at sea level (figure 30)[1]. This data is expressed in terms of altitude. The equivalent sea level oxygen concentrations have been added to the figure [(B x 0.209)/760] x 100. Purser developed an exponential equation to fit this data.

$$t_{iO_2 I} = exp\ [8.13 - 0.54\ (20.9 - \%\ O_2)] \quad \text{where } 20.9 - \%\ O_2 = \%\ \text{oxygen vitiation} \qquad (35)$$

(*in minutes*)

Dose to incapacitation = (20.9 - percent O_2) (t_i)

Speitel also adapted data from Luft, and like Purser, plotted time of useful consciousness for men at rest following sudden decompression to a range of simulated altitudes. However, Speitel accounted for the partial pressure of water in the inspired air, assuming the inspired gas has been saturated with water vapor at 37°C (P_{H_2O} = 47 mm Hg). Speitel also plotted oxygen sea level equivalent of tracheal air {or [(B-47)0.209/713] x 100} versus time of useful consciousness. The term "oxygen sea level equivalent of tracheal air" describes the oxygen exposure concentrations at sea level for which the tracheal partial pressures of O_2 match those at a given altitude. Speitel developed an exponential equation to fit this curve.



Reprinted with permission from the SFPE Handbook of Fire Protection Engineering, Copyright 1988 by the Society of Fire Protection Engineers[1]

FIGURE 30. TIME OF USEFUL CONSCIOUSNESS ON SUDDEN EXPOSURE TO HIGH ALTITUDES AND CALCULATED TIMES FOR OXYGEN SEA LEVEL EQUIVALENTS

$$t_{iO_2T} = exp\ [8.67 - 0.550\ (20.9 - \%\ O_2)] \qquad (36)$$

Note 713 mm Hg = total tracheal pressure of dry gases at sea level. Thus at sea level, the oxygen sea level equivalent of tracheal air is: (760 - 47)(0.209/713) x 100 = 20.9% O_2.

Going one step further, Speitel adapted Luft's data to account for both the partial pressure of water and CO_2. She plotted the O_2 sea level equivalent of alveolar air {[(B-87)0.209/673] x 100} vs. time of useful consciousness. Speitel developed an exponential equation to fit this curve (figure 31).

$$t_{iO_2A} = exp\ [8.55 - 0.511\ (20.9 - \%\ O_2)] \qquad \text{when } \%\ O_2 < 11.0\% \qquad (37)$$

The three curves for the inspired, tracheal, and alveolar sea level equivalents obtained from figure 30 are illustrated in figure 31.

As with exposure to HCN, time to incapacitation for exposure to low oxygen concentrations does not follow Haber's Rule since short exposures to severe hypoxia cause incapacitation very rapidly and long exposures to modest hypoxia have little effect (e.g., at 5% O_2, using equation 37: (20.9 - C)t = 15.9 x 1.53 = 24.3 percent•min, while at 10% O_2: (20.9 -C)t = 10.9 x 19.7 = 215 percent•min).

Gall et al.[61] defined Time of Useful Consciousness as the length of time between the period when the subject's oxygen supply is totally deprived (at various altitudes) and the onset of physical or mental deterioration. Gall states that at 40,000 feet useful consciousness lasts 30 seconds or less; at 35,000 feet, from 45 to 60 seconds; at 30,000 feet, from 20 to 90 seconds; and at 25,000 feet, from 2 to 3 minutes.

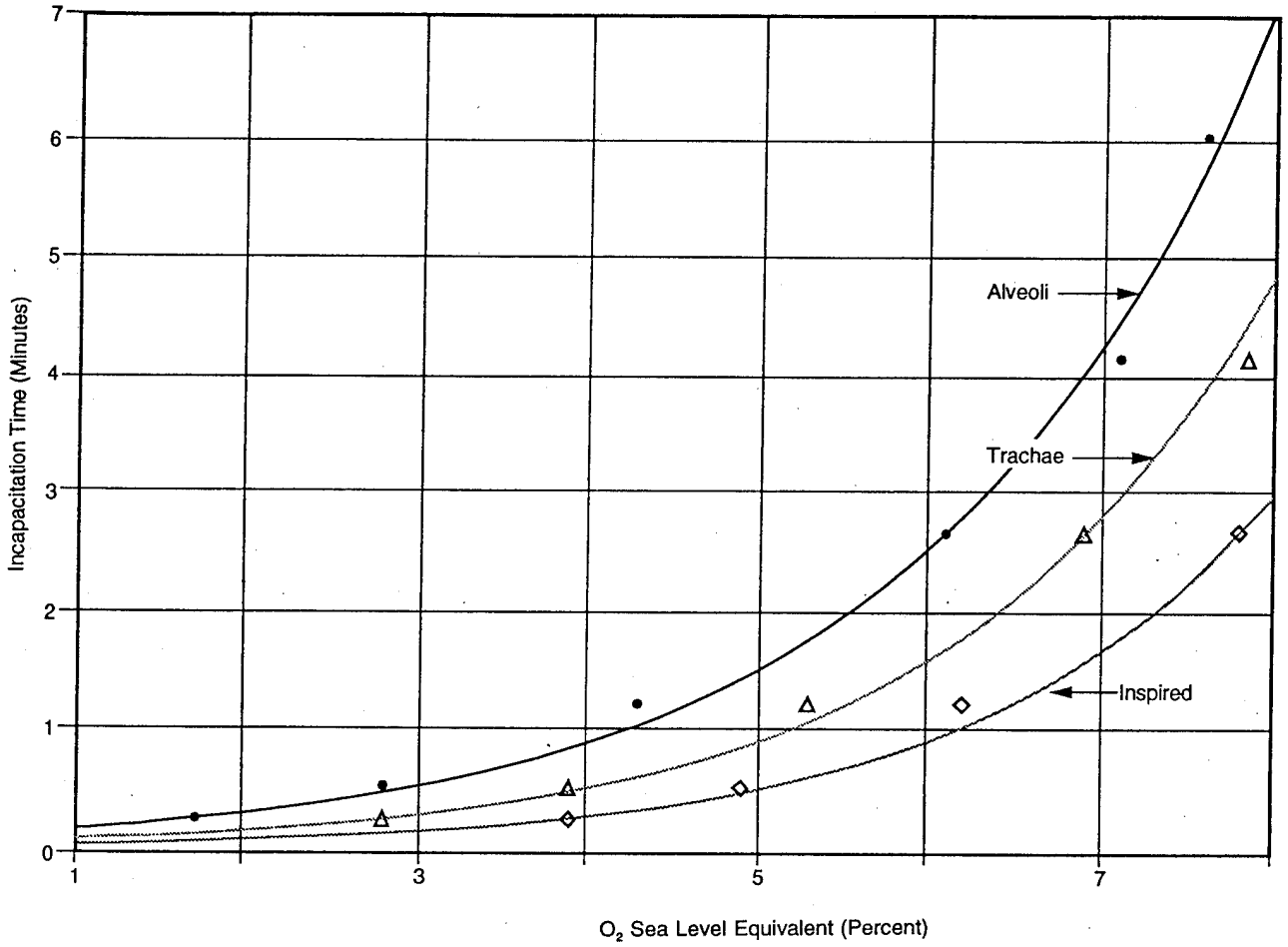
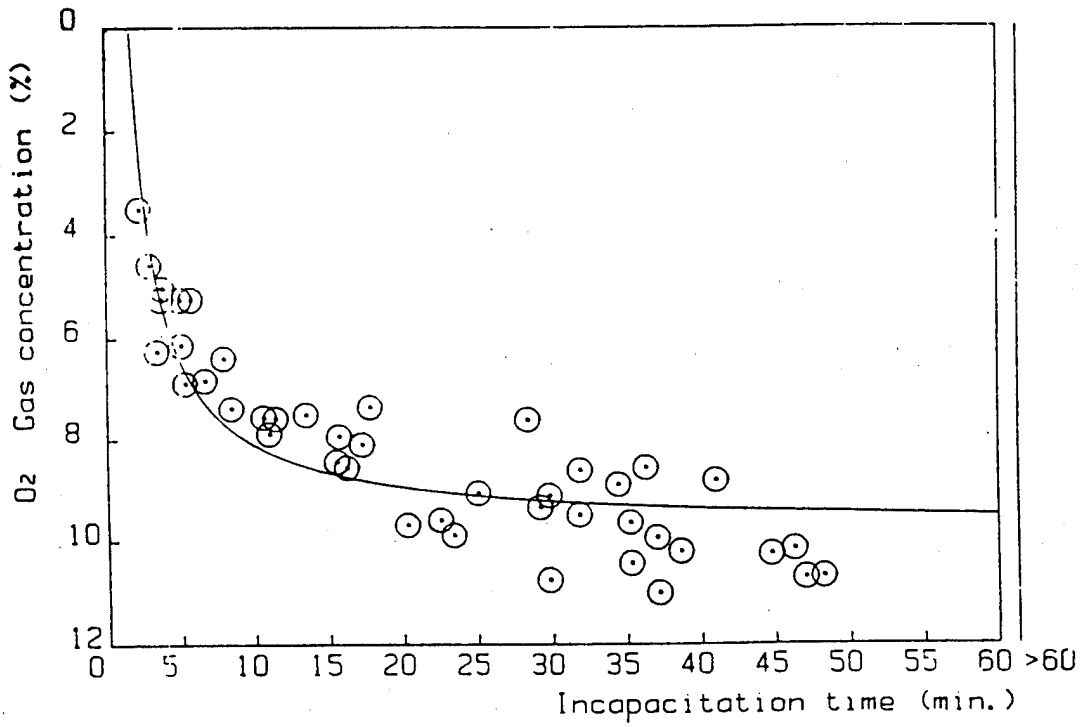


FIGURE 31. TIME TO INCAPACITATION BY HYPOXIA DERIVED FROM ALTITUDE DATA (LUFT)

Izraeli et al. determined the "Time of Useful Consciousness" (TUC) for exposures to simulated altitude of 25,000 feet in an altitude chamber. Seventeen healthy 18 to 20 year old student pilots were exposed twice to a simulated altitude of 25,000 feet. Median values of TUC were 267 seconds in the first exposure and 240 seconds in the second. Each subject had an individual oxygen mask to wear during a denitrogenation period. The ascent rate was 5000 feet min^{-1}. At 25,000 feet and 60 seconds after removal of the oxygen mask, the volunteers started the performance task. The task was to add pairs of 2-digit random numbers. Every subject received 16 pages, each containing 20 exercises. The endpoints for determination of the TUC were either two consecutive mistakes or inability of the subject to perform his task[62].

Sakurai[9] did a study where groups of 8 mice were exposed whole body in a chamber while exercising on activity wheels. The endpoint measured was incapacitation, when they ceased to run in the wheel (figure 32). Times to incapacitation are longer than those for humans (using the calculated values of oxygen sea level equivalent of alveolar air for human altitude data). Note the human data are based on decompression experiments (Luft), while the mouse studies are preformed at normal atmospheric pressure. The human data is based on men at rest, while the mouse data is based on active subjects. Sakurai obtained a regression equation to fit his data.

$$t_i = \frac{16.6}{(9.77 - C_{Oxygen})} \qquad \text{when } C_{Oxygen} < 9.77\% \qquad (38)$$



Reprinted with permission from the Journal of Fire Sciences, Copyright 1989 by Technomic Publishing Co., Inc.[9]

FIGURE 32. TIME TO INCAPACITATION FOR ACTIVE MICE EXPOSED TO LOW OXYGEN CONCENTRATIONS (SAKURAI)

Levin et al.[63] exposed rats for 30 minutes to low atmospheric concentrations of O_2. Only 1 out of 42 exposed rats died postexposure and this death occurred on day 4. The 30-minute LC_{50} value was 7.5% with 95% confidence limits of 7.3% to 7.7%.

Matijak-Schaper and Alarie[64] exposed male mice in groups of 4 to low atmospheric concentrations of oxygen for 30 minutes. The mice were placed in a glass chamber in which the animal bodies were restrained by plethysmographs and only the heads protruded into the chamber interior. The animals were essentially unaffected by a reduction of the oxygen level from 20.9% to 9.0%. Slight decreases in the average respiratory rate were observed at 9.0% oxygen. A summary of the experimental data collected for the determination of the LC_{50} for low oxygen atmospheres is presented in table 13.

The calculated 30-minute LC_{50} is 6.7%.

The above studies for the most part consisted of animals and humans at rest. An increased level of activity will most probably result in incapacitation at higher oxygen concentrations.

TABLE 13. SUMMARY OF LOW-OXYGEN LETHALITY DATA FOR MICE

Exposure Time (minutes)	% O_2	% N_2	Number of Deaths	Time to Death (minutes)
30	9.0	91	0/4	------
30	7.5	92.5	2/4	3, 12
30	6.3	93.7	3/4	2, 4, 14
30	5.2	94.8	4/4	5, 6, 11, 12

CONVECTIVE HEAT. Crane performed a literature search of human heat tolerance limits in 1978[65]. The endpoint of interest was physical collapse from thermal overload. The measured thermal parameter was air temperature, the subjects were "healthy adult males," and the clothing was "usual business dress."

Crane utilized a least squares linear regression technique to fit an equation to the four data points presented in table 14.

TABLE 14. EXPERIMENTAL VALUES OF ENDURANCE TIME AT VARIOUS ENVIRONMENTAL AIR TEMPERATURES

Air Temperature °C	Time to Collapse Minutes
50	300
105	25
120	15
200	2

Crane derived the equation:

$$t_c = Q_0/T^{3.61} \quad \text{where} \quad \begin{array}{l} t_c \text{ is time to thermal collapse, in minutes,} \\ T \text{ is air temperature, in °C, and} \\ Q_0 = 4.1 \times 10^8 \end{array} \qquad (39)$$

Q_0 is the statistically derived proportionality constant; it is a quantity related to the number of calories that the body could absorb before incapacitation. It can be used to calculate a time to collapse for a person exposed to a constant air temperature of T°C. It should be emphasized that the use of the value of $4.1 \times 10^{+8}$ for Q_0 signifies that the exposed individual is a healthy male adult dressed in a particular

way. The value of Q_0 would be different for individuals of other ages, body sizes, states of health, types of attire, etc.

Crane found four additional data points in the literature after deriving the above equation (table 15). Plotting these four new points on a graph of the original equation revealed that two fell on the original line and the remaining two were near it.

TABLE 15. ADDITIONAL EXPERIMENTAL VALUES FOR THERMAL ENDURANCE

Air Temperature °C	Time to Collapse Minutes
82	49
93	32
115	20
140	5

Data from tables 14 and 15 are illustrated in figure 33.

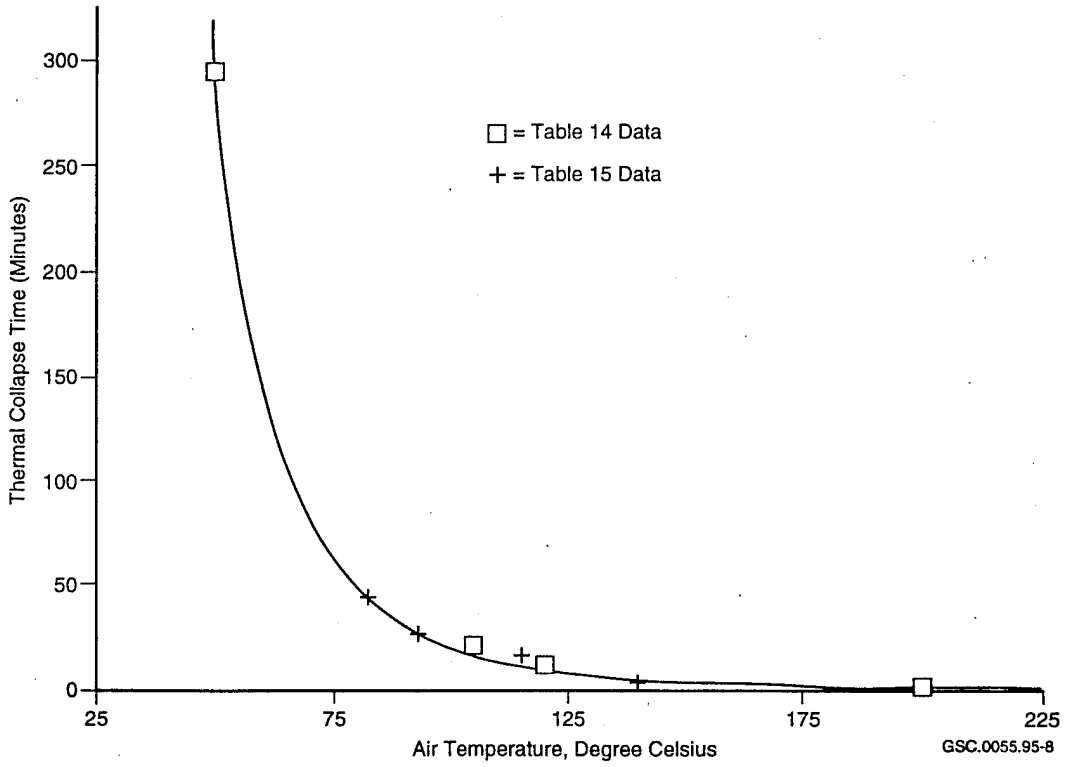


FIGURE 33. TIME OF THERMAL COLLAPSE FOR CLOTHED HUMAN MALE SUBJECTS[65]

Blockley[37,66] experimentally determined ranges of heat tolerance for unclothed human subjects at rest in dry and humid air. A plot of this data can be found in figure 34. Purser derived the following expression for time to incapacitation from this data using the average t_c for humid and dry air[1]:

$$t_{lh} \text{ (min)} = exp\ [5.1849 - 0.0273\ T(°C)] \qquad (40)$$

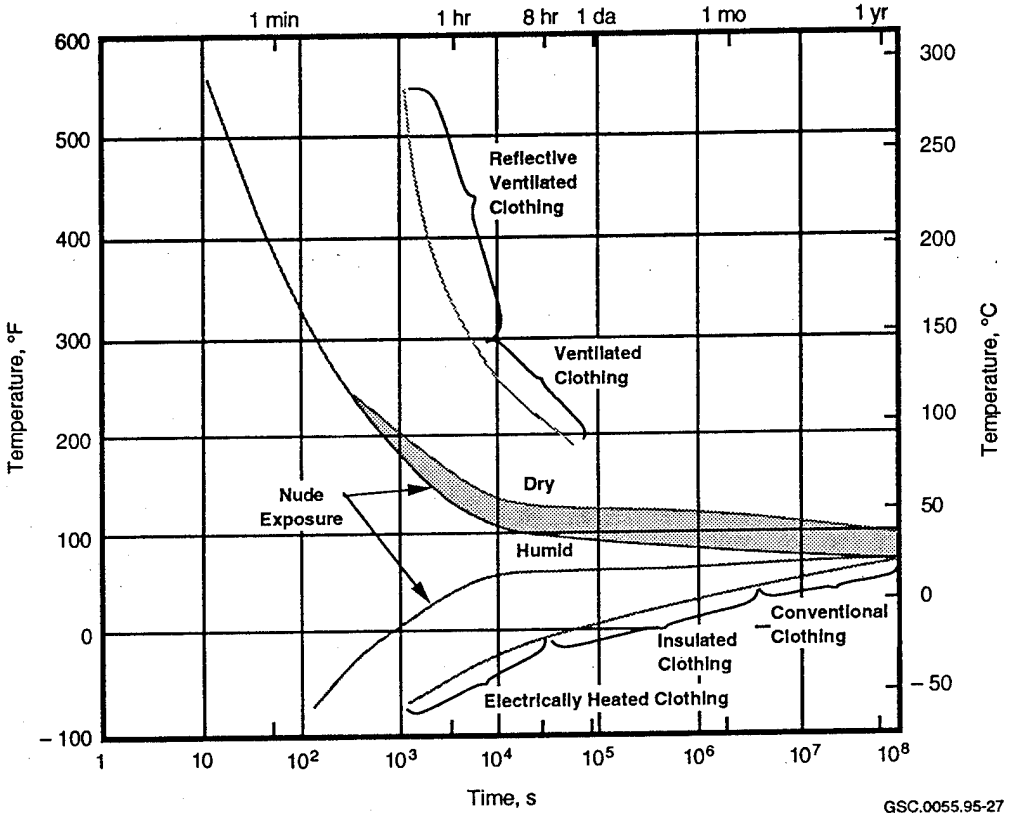


FIGURE 34. HEAT AND COLD TOLERANCE WITH AND WITHOUT PROTECTIVE CLOTHING FOR HUMAN SUBJECTS AT REST (BLOCKLEY)[37]

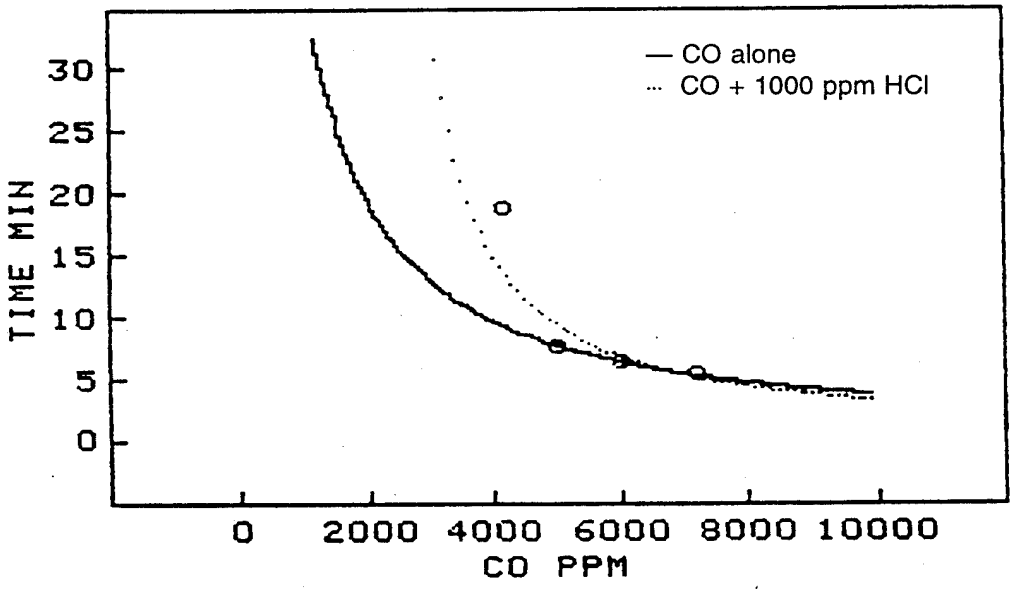
MIXED GASES.

CARBON MONOXIDE AND HYDROCHLORIC ACID. Hartzell et al. exposed 6 male Sprague-Dawley rats at a time to mixtures of CO and HCl in an exposure apparatus similar in size and shape to that commonly used in the NBS smoke toxicity test. However it was modified to create a "flow through" system for head only exposures. The air flow through the exposure chamber ranged from about 30 L/min to about 200 L/min. The LC_{50} data suggest additivity. Table 16 shows data for gas mixtures in which the summation of fractional lethal doses approximates unity for 50% postexposure lethality with 30-minute exposures and a 14-day postexposure observation period[10].

TABLE 16. SUMMATION OF FRACTIONAL EFFECTIVE (LETHAL) DOSES FOR 30-MINUTE EXPOSURE OF RATS TO MIXTURES OF CARBON MONOXIDE AND HYDROGEN CHLORIDE

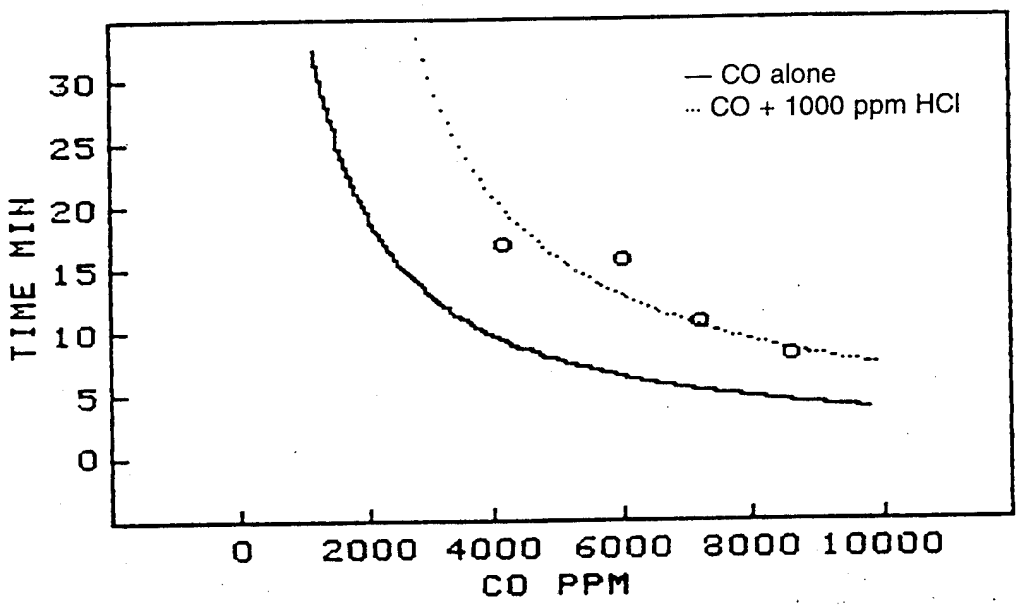
CO		HCl		
ppm	Fractional Lethal Dose	ppm	Fractional Lethal Dose	Σ FED (50% Lethality)
6400	1.0	----	----	1.0
5700	0.89	600	0.16	1.05
5300	0.83	1000	0.26	1.09
4150	0.65	1900	0.50	1.15
3000	0.49	2100	0.55	1.04
----	----	3800	1.0	1.0

In the same paper, Hartzell et al. showed that the overall effect of the presence of HCl on incapacitation of rats by CO is relatively minor. A beneficial effect is observed in a "window" of perhaps 400 to 1000 ppm HCl and with CO concentrations of up to about 4000 ppm (figures 35 and 36). The authors emphasized that the effects of a sensory irritation on CO toxicity may be limited to rodents.



Reprinted with permission from the Journal of Fire Sciences, Copyright 1987 by Technomic Publishing Co., Inc.[10]

FIGURE 35. CONCENTRATION VERSUS TIME TO INCAPACITATION FOR CARBON MONOXIDE ALONE AND IN THE PRESENCE OF 1000 PPM HYDROGEN CHLORIDE



Reprinted with permission from the Journal of Fire Sciences, Copyright 1987 by Technomic Publishing Co., Inc.[10]

FIGURE 36. CONCENTRATION VERSUS TIME TO INCAPACITATION FOR CARBON MONOXIDE ALONE AND IN THE PRESENCE OF 1000 PPM HYDROGEN CHLORIDE AS CONTAINED IN NONFLAMING PVC SMOKE

There is no primate data on HCl and CO mixtures. However, it has been shown by Kaplan et al.[18] that HCl increases the RMV in primates (discussed on page 16, see figure 14). This increased uptake of other gases may increase the toxic effect.

CARBON MONOXIDE AND HYDROGEN CYANIDE. Whether the effects of CO and HCN, when inhaled as a mixture, are additive or synergistic have been the subject of some research and considerable speculation over at least the past 60 years. Hoefer[67], in 1926, reported exposing cats to such mixtures and measuring the exposure time required to produce paralysis. A recent critique of 11 such studies was published by Tsuchiya[68]. The conclusions reached by the individual authors were almost equally divided for and against a synergistic combined effect. Tsuchiya attributed these differing conclusions to one or more of the following deficiencies: (a) improper use of the term synergism, (b) conclusions by the authors that were inconsistent with the experimental data, or (c) valid statistical treatments were not applied to the experimental data.

Crane et al. have shown that the combined effect of CO and HCN is less than additive. Crane et al. exposed 63 rats, one at a time, to defined mixtures of CO and HCN in an exposure chamber consisting of a shock grid and a motor-driven drum controlled at 6 cm/sec. For the concentrations studied, it appears that CO was contributing an undiminished potency to the combined effect while the HCN was only about two-thirds as effective as when administered alone[7]. The concentrations of gas mixtures studied ranged from:

84 ppm HCN to 215 ppm HCN
1332 ppm CO to 6385 ppm CO

Hartzell et al. in contrast, have shown that CO and HCN appear to be additive when expressed as a fractional dose required to cause an effect (incapacitation and lethality)[69,70].

Levin et al. in 1986 conducted 10-, 20-, and 60-minute rat exposures of mixtures of CO and HCN and concluded the LC_{50}s of these gases are additive. These data are listed in table 17[11].

TABLE 17. ADDITIVITY OF CARBON MONOXIDE AND HYDROGEN CYANIDE LC_{50}s (LEVIN'S 1986 DATA)

Exposure Time (Minutes)	Percent of LC_{50}			Number of Rats	
	CO	HCN	Total	Died	Tested
10	52	47	99	3	6
	52	47	99	2	6
20	51	60	111	4	6
	50	57	107	4	6
60	51	56	107	2	6
	53	56	109	4	6

ANY GAS AND CARBON DIOXIDE (EFFECT OF CO_2 ON RMV). Although carbon dioxide is not toxic to man at concentrations of up to 5%, it stimulates breathing so that at 3% the RMV is approximately doubled; at 5%, tripled; and at 7%, the RMV increases five-fold[12]. The ventilatory response to carbon dioxide varies among individuals and reported data also vary. An average curve was constructed by Purser[1] from data given in three sources[40,41,42] and is shown in figure 37. Purser derived the following regression equation for this curve.

$$RMV\ (L/\text{min}) = exp\ [0.2496\ x\ \%\ CO_2 + 1.9096] \tag{41}$$

Purser calculated a multiplication factor (V_{CO_2}) for the enhanced uptake of other narcotic gases.

$$V_{CO_2} = \frac{exp\ (0.2496\ x\ \%\ CO_2 + 1.9086)}{6.8} \quad \text{where 6.8 L/min is the resting RMV} \tag{42}$$

Using equation 42, the value for V_{CO_2} at 5% CO_2 is 3.45 and the value for V_{CO_2} at 10% CO_2 is 12.0.

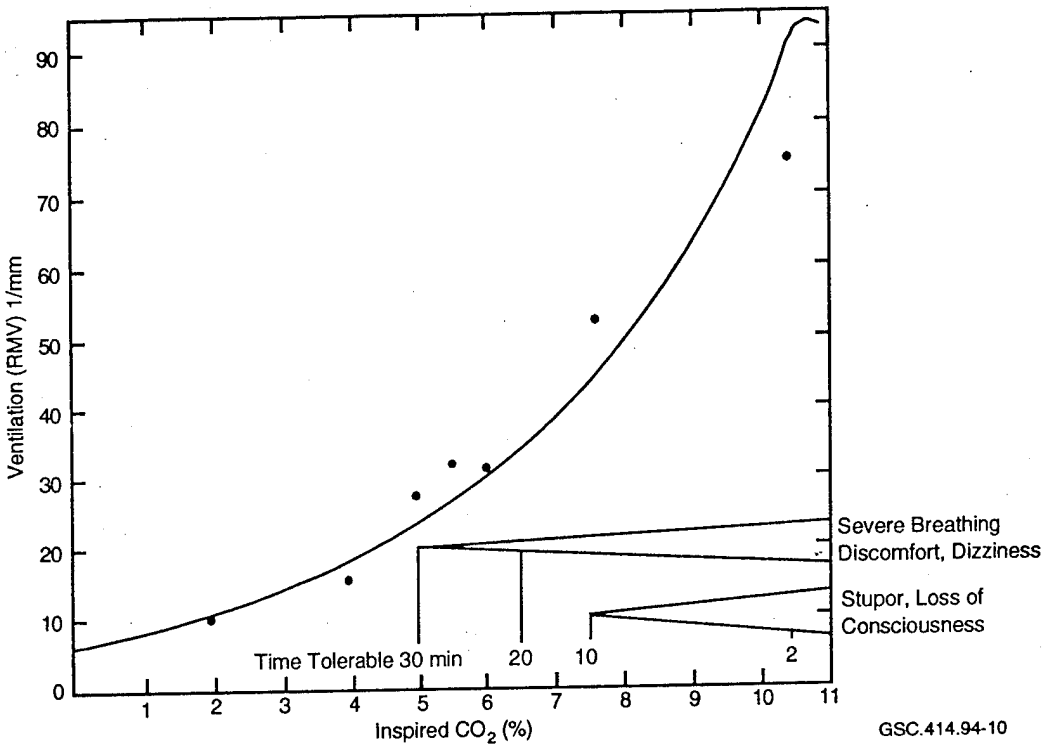


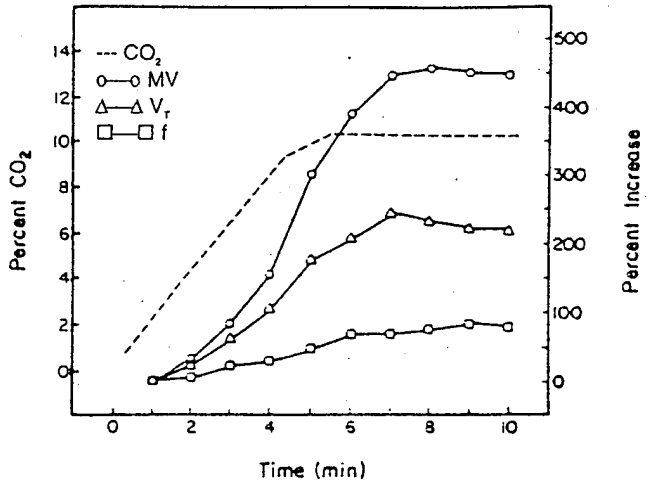
FIGURE 37. EFFECT OF CARBON DIOXIDE ON HUMAN VENTILATION

This hyperventilation is stressful to the subject, and it can increase the rate of uptake of other toxic gases and decrease the time to effect. The uptake rate however is not necessarily increased by the same factor as the increase in RMV. Body uptake may be limited due to gas solubilities and saturation at the alveoli. Experimental data on combination exposures are limited. It may be safe to assume that the strongly soluble acid gases, the main toxic effect being destruction of the respiratory system, would dissolve quantitatively onto these tissues, such that the effective dose would increase by the same factor as the increase in RMV (assuming these irritant gases wouldn't effect the respiratory response).

The effect of high carbon dioxide concentrations on RMV appears to be markedly different for different species. Kaplan et al.[18] performed a 10-minute CO_2 challenge test on three anesthetized baboons. The change in the respiratory parameters as a percentage of baseline values is plotted in figure 38. It can be seen that at 10% CO_2 the RMV is 5.5 times the baseline (far less than predicted by Purser's RMV curve for humans).

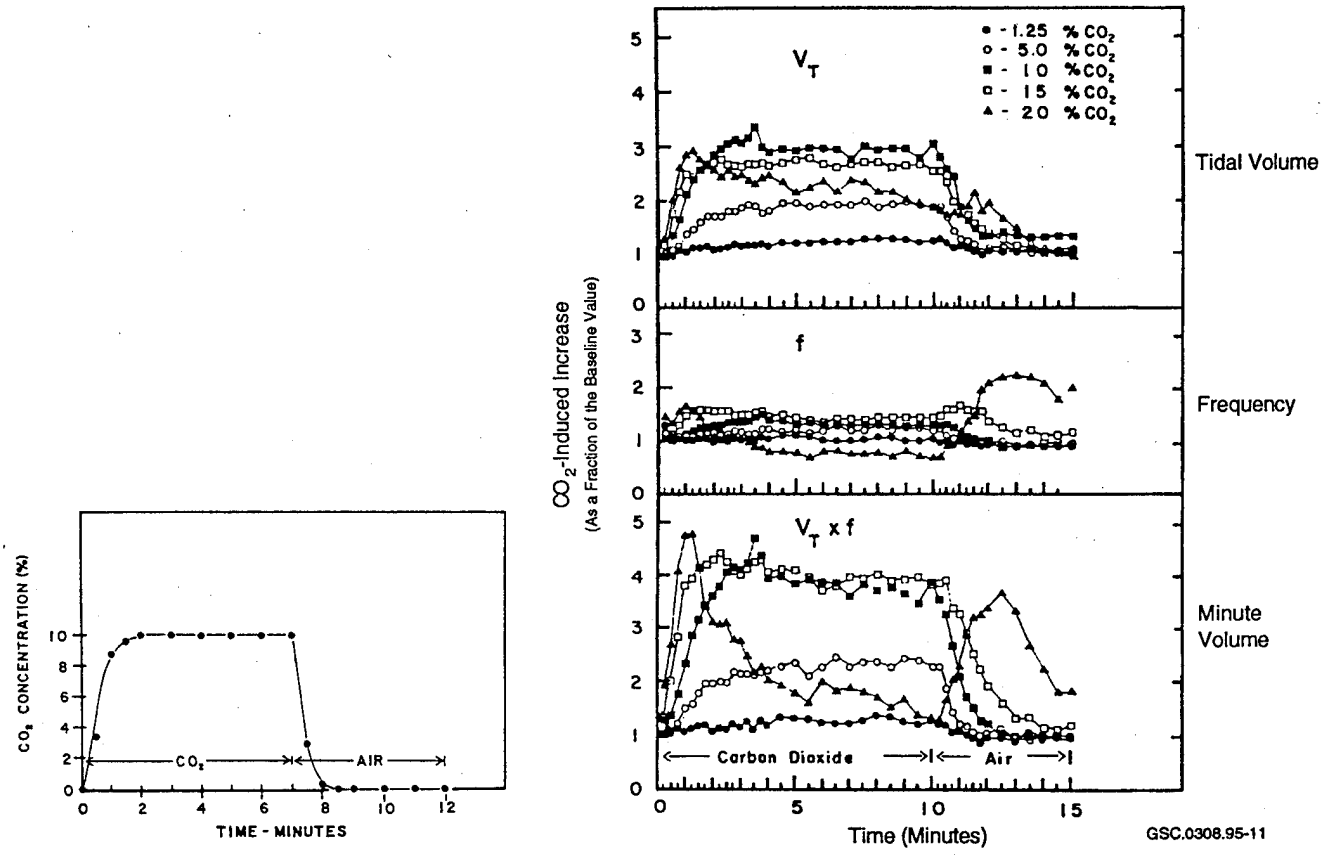
Wong and Alarie[71] measured the tidal volume (V_T) and the respiratory frequency (f) and calculated the respiratory minute volume (V_T x f) for unanesthetized, unrestrained guinea pigs. All gas mixtures contained 20% O_2 with an N_2 balance. At 10% and 15% CO_2 the RMV is 4 times the baseline RMV. At 5% CO_2 the RMV is 2.2 times the baseline RMV. This data is plotted in figure 39.

Chapin[72] determined the increase in RMV for unrestrained and unanesthetized hamsters exposed to CO_2 concentrations up to 35%. The peak ventilation was found when the animals breathed about 20% CO_2 in 20% O_2 and 60% N_2. At 5% CO_2 the RMV is 2 times the baseline RMV. At 10% CO_2 the RMV is about 3 times the baseline. At 15% CO_2 the RMV is 5 times the baseline, and at 20% CO_2 the RMV is about 6.5 times the baseline.



Reprinted with permission from the Journal of Toxicology and Environmental Health, Copyright 1988 by Hemisphere Publishing Co., New York.[18]

FIGURE 38. CHANGE IN RESPIRATORY PARAMETERS OF THE ANESTHETIZED BABOON AS A PERCENT OF BASELINE VALUES DURING THE 10-MINUTE CARBON DIOXIDE CHALLENGE TEST



Reprinted with permission from Toxicology and Applied Pharmacology, Copyright 1982 by Academic Press.[71]

FIGURE 39. EFFECT OF CARBON DIOXIDE ON PULMONARY PERFORMANCE IN UNANESTHETIZED, UNRESTRAINED GUINEA PIGS (WONG AND ALARIE)

Lai et al.[73] exposed rats to 5% or 7% CO_2 in air for up to 3 weeks. The average ventilation, $\dot{V}_E$, (the product of V_T and f) of the 28 rats exposed to 5% CO_2 increased to 215.9 ± 53.8% of control at 2 hours (a 2.16-fold increase). The 12 rats receiving 7% CO_2, the $\dot{V}_E$ increased to 213.5 ± 46.8% of control at 2 hours. The ventilatory parameters (V_T, f, $\dot{V}_E$) as well as the rate of O_2 uptake $\dot{V}_{O_2}$, are plotted versus time in figures 40 and 41. The rate of uptake of O_2 increased to 1.3 times the baseline uptake rate at 2 hours into the exposure. The oxygen uptake increased in the presence of 5% CO_2 but not by as great a factor as the RMV increase (1.3 versus 2.2).

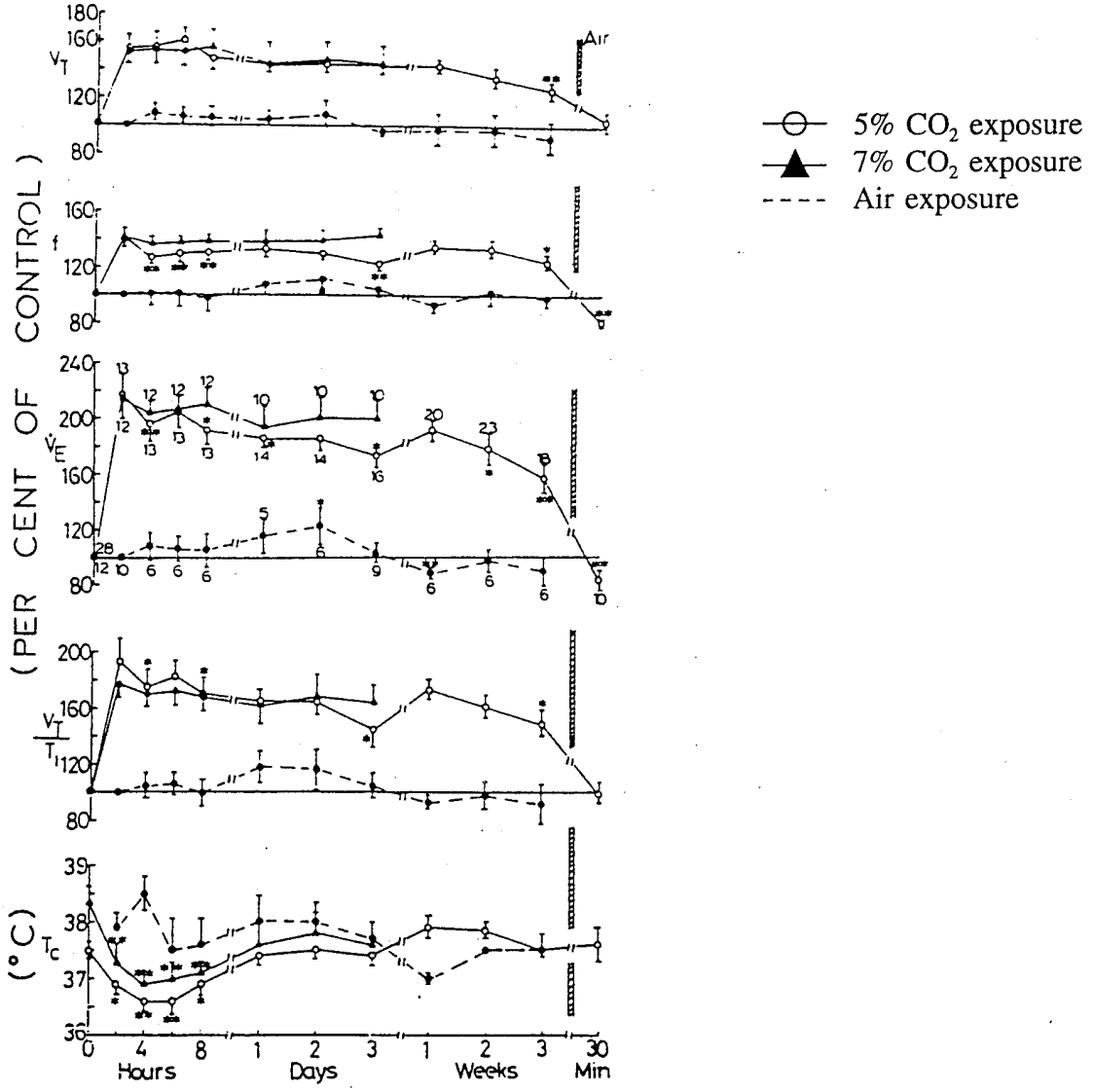
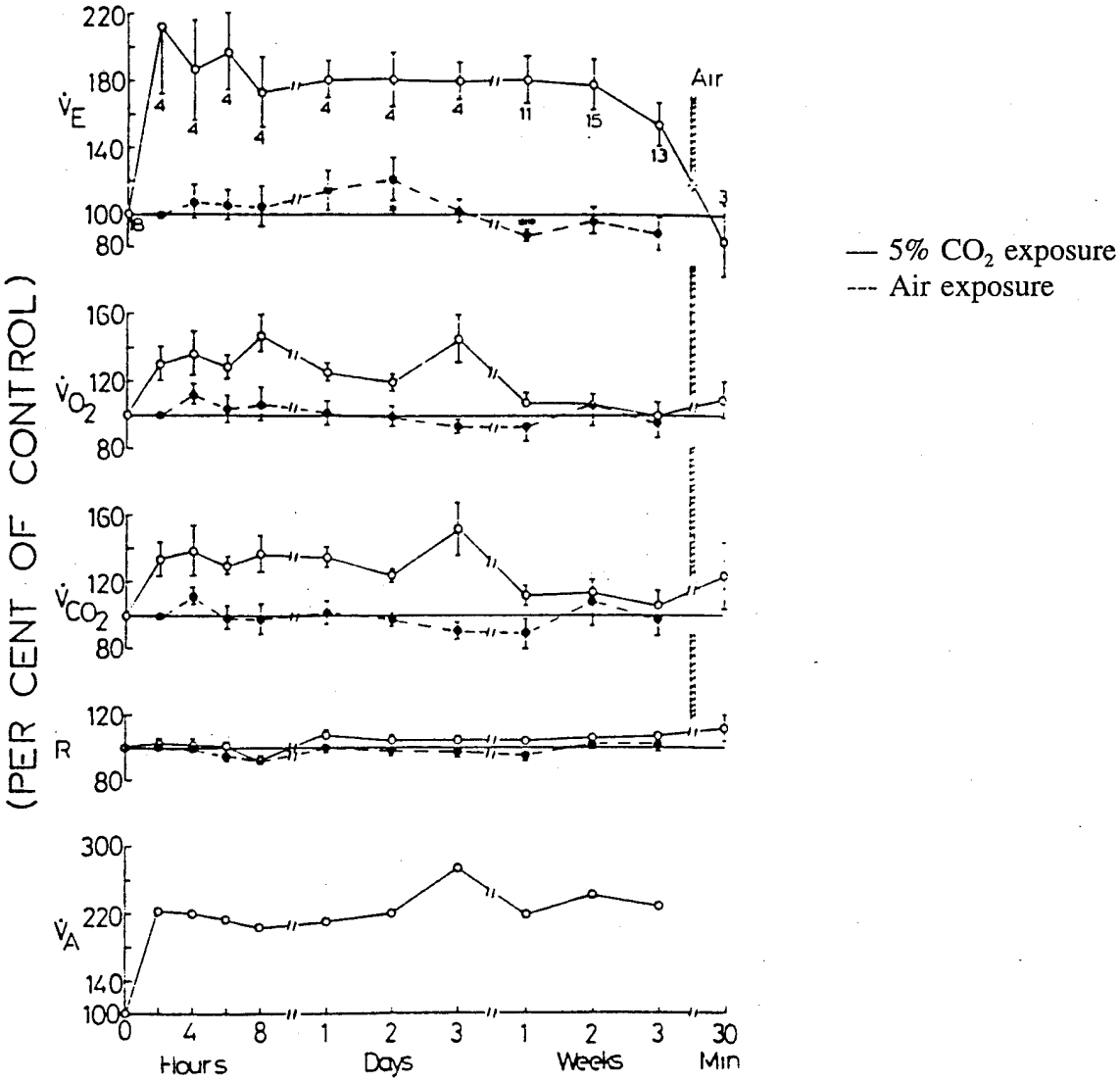


FIGURE 40. VENTILATORY PARAMETERS (V_T, f, $\dot{V}_E$, AND V_T/T_I) PER 100 g BODY WEIGHT, EXPRESSED AS A PERCENTAGE OF BASELINE (AIR BREATHING AT BEGINNING OF STUDY) AND BODY TEMPERATURE (T_c) DURING CONTINUOUS 5% AND 7% CARBON DIOXIDE EXPOSURE AND AIR BREATHING (LAI)



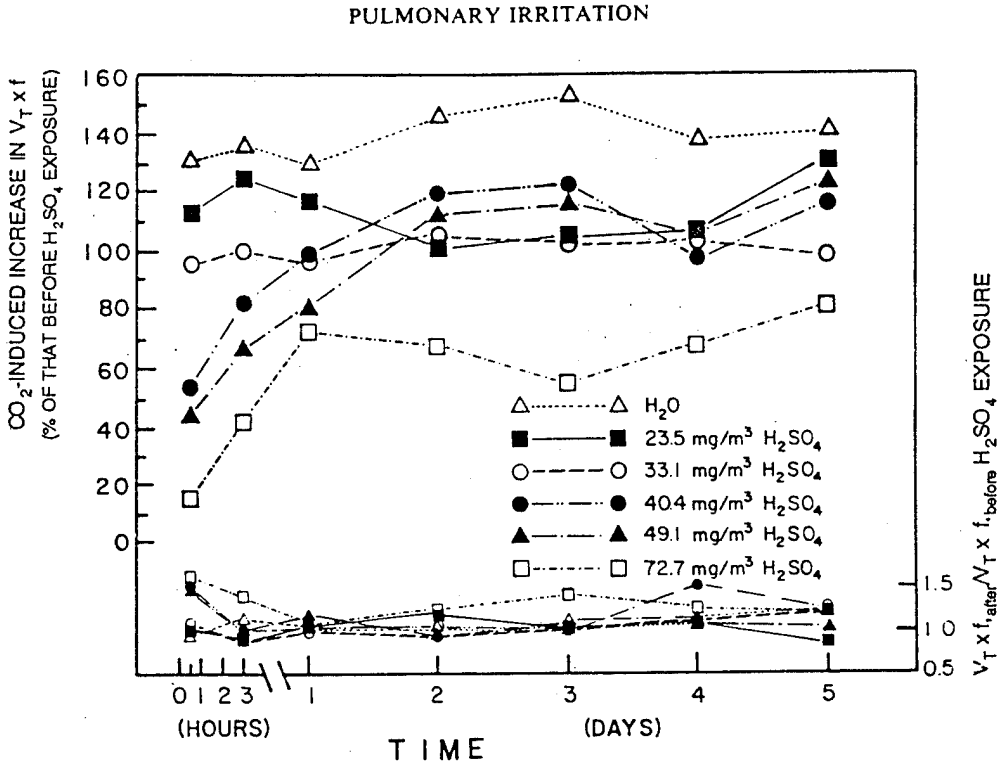
Reprinted with permission from the Journal of Applied Physiology, Copyright 1981 by The American Physiological Society.[73]

FIGURE 41. $\dot{V}_E$, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, R, AND ESTIMATED $\dot{V}_A$, PER 100 g BODY WEIGHT, EXPRESSED AS A PERCENTAGE OF CONTROL IN RATS EXPOSED TO 5% CARBON DIOXIDE OR AIR

Wong and Alarie have shown that sulfuric acid mist reduces the CO_2 induced increase in minute volume in guinea pigs (figure 42[71]).

CARBON MONOXIDE AND CARBON DIOXIDE. Levin[74] reported a synergistic effect of CO and CO_2 and claimed that the synergistic effect for rats was as much as a factor of two. She explained that the presence of relatively high concentrations of CO_2 stimulates respiration and increases the uptake of CO, resulting in a shorter survival time.

More recently, Levin et al. determined the LC_{50} values of CO for exposures of rats for 5, 10, 20, 30, and 60 minutes in the presence or absence of approximately 5% CO_2. With the exception of the 5-minute case, the LC_{50} values were statistically significantly lower (based on the 95% confidence limits) in the presence of 5% CO_2 than its absence (table 18, reference [11]).



Reprinted with permission from the Toxicology and Applied Pharmacology, Copyright 1982 by Academic Press.[71]

FIGURE 42. THE EFFECT OF SULFURIC ACID ON BASELINE MINUTE VOLUME V_T•f (SMALL SYMBOLS) AND ON THE CARBON DIOXIDE-INDUCED INCREASE IN V_T•f (LARGE SYMBOLS) IN THE GUINEA PIG

TABLE 18. LC_{50} VALUES FOR CARBON MONOXIDE ALONE AND CARBON MONOXIDE PLUS CARBON DIOXIDE (LEVIN'S 1986 DATA)

Exposure Time (Minutes)	CO (ppm)		CO + 5% CO_2 (ppm)	
5	14,000	(12,000-16,000)	11,700	(10,800-12,700)
10	9800	(9200-10,500)	7800	(7500-8200)
20	7400	(7100-7700)	5600	(5500-5800)
30	6600	(6100-7300)	3900	(3400-4500)
60	4900	(4600-5200)	3100	(2800-3400)

Levin's rat LC_{50} data for CO exposures with and without 5% CO_2 (table 18) provides a basis to predict a multiplication factor for apparent enhanced uptake of CO due to presence of 5% CO_2. This multiplication factor $(mf)_{5\%\ CO_2+\ CO}$ increases as the exposure time increases and as the CO concentration decreases. The multiplication factor ranges from 1.2 for a 5-minute exposure to 1.6 for a 60-minute exposure to $LC_{50\ CO+5\%\ CO_2}$ concentrations.

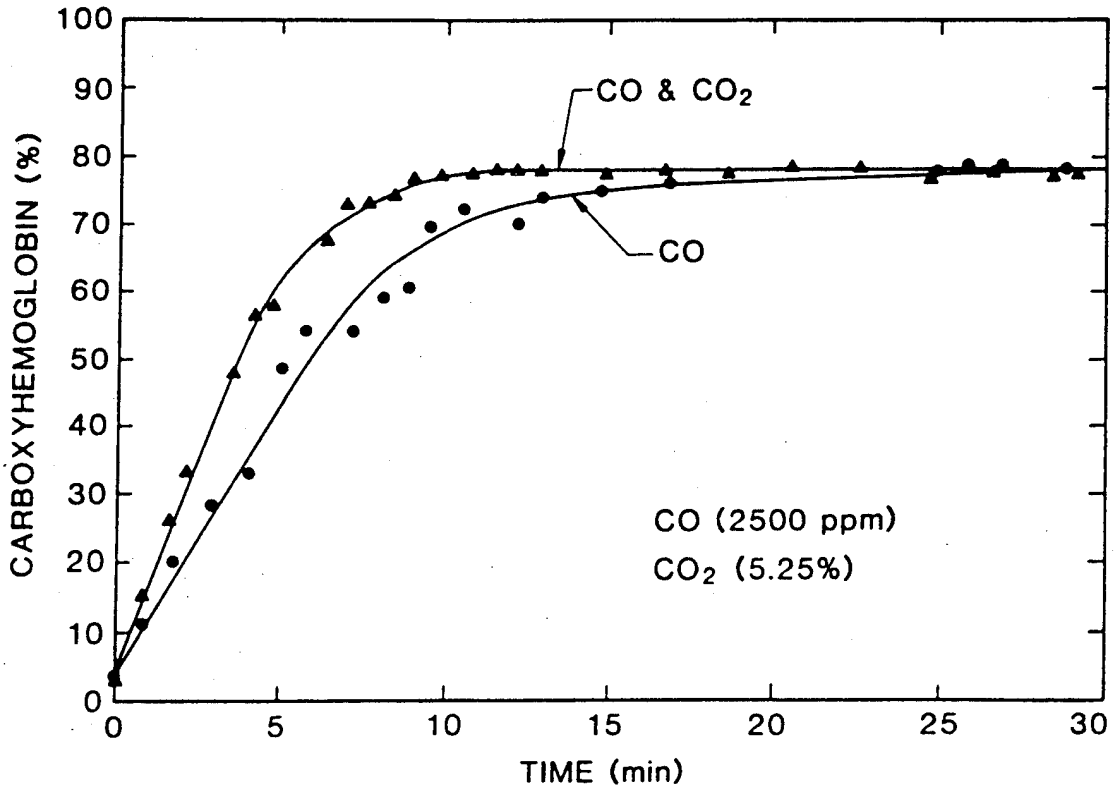
TABLE 19. MULTIPLICATION FACTORS FOR APPARENT ENHANCED UPTAKE OF CARBON MONOXIDE DUE TO THE PRESENCE OF 5% CARBON DIOXIDE FOR RATS

$t_{exposure}$ (Minutes)	$LC_{50\ CO}$ (ppm)	$LC_{50\ CO+5\%\ CO_2}$ (ppm)	$\frac{LC_{50\ CO}}{LC_{50\ CO\ +\ 5\%\ CO_2}} = (mf)_{5\%\ CO_2+\ CO}$
5	14,000	11,700	1.20
10	9800	7800	1.26
20	7400	5600	1.32
30	6600	3900	1.69
60	4900	3100	1.58

In an earlier study, Levin et al.[52] studied the arterial blood of restrained rats exposed to combinations of CO and CO_2. Six rats were exposed to 2500 ppm CO with and without 5.25% CO_2 (figure 43). A CO concentration of 2500 ppm was selected as it is the lowest lethal level of CO in the presence of 5.25% CO_2 for restrained rats. The calculation of the slopes of the first seven values in each of the curves shown in figure 43 indicates that the initial rate of formation of COHb was 1.5 times greater from 2500 ppm CO in the presence of 5.25% CO_2 than in the absence of CO_2. The COHb equilibrium level was the same (78%).

$$\frac{rate\ of\ COHb\ formation\ CO\ +\ 5\%\ CO_2}{rate\ of\ COHb\ formation\ CO} = 1.5 \qquad (43)$$

The value for the increase in RMV for rats in the presence of 5% CO_2 (V_{CO_2}) was shown to be 2.2 by Lai et al. The value for the increase in RMV for guinea pigs in the presence of 5% CO_2 was shown by Wong and Alarie to be 2.2. It appears that 5% CO_2 increases the uptake rate of CO but does not match the CO_2 induced increase in RMV (V_{CO_2}) in rats.



Reprinted with permission from Toxicology, Copyright 1987 by Elsevier Scientific Publishers Ireland Ltd.[52]

FIGURE 43. RATE OF CARBOXYHEMOGLOBIN FORMATION FOR RESTRAINED RATS DURING EXPOSURE TO 2500 PPM CARBON MONOXIDE ALONE OR WITH 5.25% CARBON DIOXIDE[52]

Crane[75] exposed active rats to CO concentrations ranging from 5,500 to 14,000 ppm with or without added CO_2 concentrations of 8% and 10.4%. There was no change in the times to incapacitation or the times to death resulting from the addition of CO_2. Aware of Levin's early work, Crane repeated his own experiments with mixtures of 5,000 ppm CO and from 4% to 13% CO_2, and again there was no significant change in effective times. A possible explanation for the apparent disparity between Crane and Levin's data was that the CO_2 induced increase in RMV may be greater for restrained rats than for active rats.

NITROGEN DIOXIDE AND CARBON DIOXIDE. Levin et al.[32] exposed rats for 30 minutes to approximately 200 ppm NO_2 with or without 5% CO_2. All exposures were conducted using the chemical analysis system and the animal exposure system that was designed for the NBS Toxicity Test Method. The NBS Toxicity Test Method consists of a 200 liter rectangular chamber with 6 animal ports. The animal heads were inserted into the chamber after steady state gas concentrations were achieved. The LC_{50} value for NO_2, based on a 54 animal study, was 200 ppm with 95% confidence limits of 190 to 210 ppm. All deaths from NO_2 exposures occurred during the postexposure period between 90 minutes and 24 hours. The 30-minute LC_{50} value for NO_2 in the presence of 5% CO_2, based on a 54 animal study, decreased to 90 ppm with 95% confidence limits of 70 to 120 ppm. The multiplication factor for the apparent enhanced uptake of NO_2 due to the presence of 5% CO_2 for 30-minute exposures is calculated to be 2.22.

$$(mf)_{NO_2+5\%\ CO_2} = \frac{LC_{50\ NO_2}}{LC_{50\ NO_2\ +\ 5\%\ CO_2}} = \frac{200}{90} = 2.22 \qquad (44)$$

It appears that 5% CO_2 increases the rate of uptake of NO_2 to match the CO_2 induced increase in RMV in rats (2.22 vs. 2.2). NO_2 is soluble in water. This solubility probably accounts for the quantitative uptake of NO_2.

The study above was for restrained rats. It is not known if this multiplicative effect would occur for active rats (e.g., would it occur for rats whose RMV is already double their resting RMV?).

There is no experimental data on humans or primates to determine if 5% CO_2 results in a decrease in incapacitative or lethal concentrations of NO_2 as predicted by Purser's RMV equation for increased uptake in the presence of CO_2. Purser's RMV equation for humans predicts an increase in RMV by a multiplication factor (V_{CO_2}) of 3.45 in the presence of 5% CO_2. Thus it is a reasonable assumption that the multiplication factor for the enhanced uptake of NO_2 in the presence of CO_2 would match the V_{CO_2} in humans.

REDUCED OXYGEN AND CARBON DIOXIDE. Carbon dioxide has been shown to have a marked beneficial effect on resistance to incapacitation for low oxygen. This is partly due to the hyperventilatory effect that increases the rate of oxygen uptake and partly due to the rightward shift in the oxygen dissociation curve due to the lowering of the blood pH resulting in the liberation of more oxygen from the oxyhemoglobin[76,77].

Karl et al.[77] exposed eight conscious rhesus monkeys to normal (21%) O_2 and hypoxic (12%, 10%, and 8%) O_2, all with nitrogen balance breathing atmospheres with and without the addition of 5% CO_2. The animals were trained to perform a lever press. Each exposure session lasted 90 minutes: 30-minute baseline (21% O_2), 30-minute experimental gas mixture, and 30-minute recovery (21% O_2). The cerebral P_{O_2} was elevated with the addition of 5% CO_2. These data are summarized in table 20.

It can be seen that the CO_2 benefit increases as the inspired oxygen concentration decreases. At 8% O_2, arterial P_{O_2} increased to 2.62 times the baseline (no CO_2 present). This approaches the RMV increase (due to the presence of 5% CO_2) for monkeys of 3.

CARBON MONOXIDE AND REDUCED OXYGEN. The most likely interaction between CO and low-oxygen hypoxia would be additive since both have the effect of reducing the percentage oxygen saturation of arterial blood, and CO also impairs the delivery of oxygen to the tissue by causing a leftward shift of the oxygen dissociation curve[1,78]. Evidence for an additive effect in mice is shown from the work of Sakurai[9]. Mice were exposed to CO + 10% O_2 and also to CO + 15% O_2. Incapacitation was found to occur earlier than expected for CO alone.

HYDROGEN CYANIDE AND REDUCED OXYGEN. Espisito and Alarie[79] exposed restrained mice to mixtures of HCN (80-106 ppm) and low oxygen (13% to 15%) for 30 minutes to determine the 30-

minute LC_{50} of the combined gases. The LC_{50} for HCN was reduced 40% from 177 ppm for HCN exposures in 21% oxygen to 89 ppm in the presence of low oxygen.

TABLE 20. CEREBRAL P_{O_2} RESPONSE TO GRADED HYPOXIA IN RHESUS MONKEYS WITH AND WITHOUT 5% CARBON DIOXIDE (KARL)

O_2 Content of Inspired Atmosphere (%)		Mean Cerebral P_{O_2} (mm Hg)		CO_2 Induced P_{O_2} Increase (as a fraction of baseline value)
		No Additional CO_2	5% CO_2 Added	
21	Pretreatment	131	93	$0.979\left(\frac{131}{93}\right) = 1.38$
	Test	145	142	
12	Pretreatment	146	125	$1.68\left(\frac{146}{125}\right) = 1.96$
	Test	70	118	
10	Pretreatment	111	89	$1.61\left(\frac{111}{89}\right) = 2.01$
	Test	28	45	
8	Pretreatment	118	137	$3.04\left(\frac{118}{137}\right) = 2.62$
	Test	22	67	

OXYGEN UPTAKE AND VENTILATION.

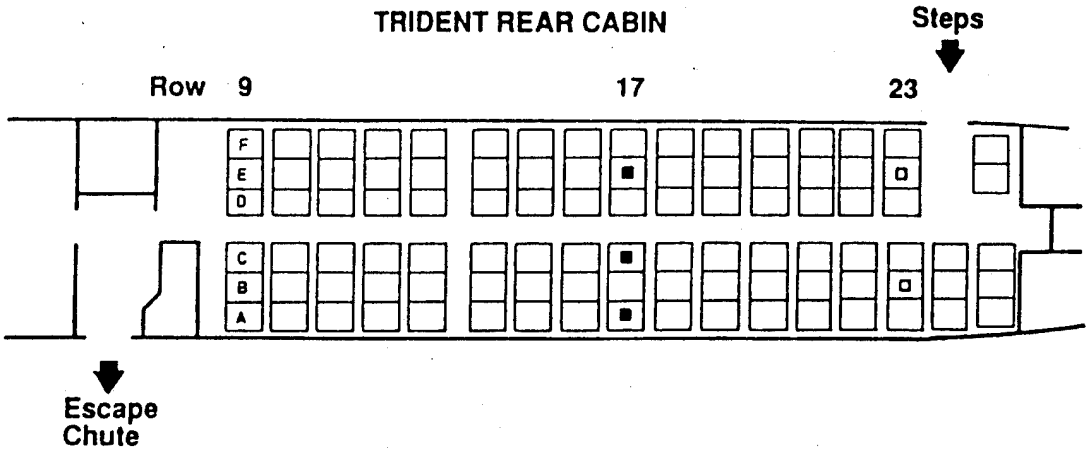
A study by Ross et al.[80] determined maximum expected respiratory minute volumes during escape from a Trident aircraft. Ventilation and oxygen consumption were quantified. Fourteen male subjects, aged 17 to 38 years, were studied under two conditions:

1. Row 17 Evacuations: The mass evacuation involved 40 volunteers occupying seven rows of seats by the rear of the cabin (rows 17 to 23). During each test, measurements were made on three subjects seated at the front of the group in row 17 in seats A, C, and E (figure 44). The other 37 subjects were present to merely simulate the crowded conditions of an actual evacuation and escaped at the same time as the monitored individuals. The individuals in row 17 had eight rows of seats to push flat to be able to escape down the port escape chute.

2. Row 23 Evacuations: These evacuations involved only two volunteers seated in row 23, seats B and E. The subjects had 14 seats to push flat and scramble. The escape was designed to produce a maximum workload.

The minute ventilation (V_I) increased by the same factor as the oxygen consumption increased (escape task vs. rest) for row 17 as well as row 23 evacuations as shown in table 21.

The units of minute ventilation are given in standard temperature and pressure, dry (STPD). Standard temperature is 0° Celsius and standard pressure is 760 mm Hg.



Reprinted with permission from Ergonomics, Copyright 1990 by Taylor and Francis, Ltd.[80]

FIGURE 44. REAR CABIN OF THE TRIDENT AIRCRAFT SHOWING THE POSITION OF SUBJECTS BEFORE ROW 17 AND ROW 23 EVACUATIONS

TABLE 21. INSPIRED MINUTE VENTILATION AND OXYGEN CONSUMPTION BEFORE AND DURING EVACUATIONS

	Row 17			Row 23		
	Rest		Escape	Rest		Escape
Mean V_I (l min^{-1} STPD)	16.72		38.99	16.71		37.35
Mean V_{O_2} (l min^{-1} STPD)	0.37		0.88	0.41		0.88
$V_{I(escape)} / V_{I\ (Rest)}$		2.33			2.24	
$V_{O_2(escape)} / V_{O_2(Rest)}$		2.37			2.15	

RELATIONSHIP BETWEEN INCAPACITATING CONCENTRATIONS AND ACTIVITY LEVELS (FOR RATS).

The incapacitating concentrations of CO and HCN can be seen to decrease with increasing activity level. However from the predictive equations of Hartzell (restrained rat) and Crane (active rat), it can be seen that the incapacitative concentration is 1.4 times greater for the restrained rat than the active rat using Crane's activity protocol. This factor holds for t_i's ranging from 5 to 60 minutes for CO and HCN (tables 22 and table 23).

TABLE 22. COMPARISON OF INCAPACITATING CARBON MONOXIDE CONCENTRATIONS FOR RESTRAINED AND ACTIVE RATS

	Calculated Concentration (ppm)		
t_i (min)	Restrained $\frac{36{,}509}{t_i} + 233$	Active $\frac{25{,}017}{t_i - 0.3} + 225$	$\frac{C_i \text{ restrained}}{C_i \text{ active}}$
5	7534.8	5547.8	1.358
10	3883.9	2804.1	1.385
20	2058.5	1494.9	1.377
30	1450.0	1067.3	1.359
60	841.5	644.0	1.307 avg. = 1.36

TABLE 23. COMPARISON OF INCAPACITATING HYDROGEN CYANIDE CONCENTRATIONS FOR RESTRAINED AND ACTIVE RATS

	Calculated Concentration (ppm)		
t_i (min)	Restrained $\frac{698}{t_i} + 92$	Active $\frac{564}{t_i} + 63$	$\frac{C_i \text{ restrained}}{C_i \text{ active}}$
5	231.6	175.8	1.317
10	161.8	119.4	1.355
20	126.9	91.2	1.391
30	115.2	81.8	1.408
60	103.6	72.4	1.431 avg. = 1.38

It has not been shown if this relationship holds for the acid gases due to lack of t_i data for restrained rats. Likewise, it is not known if this relationship holds for LC_{50}s for acid gases due to the lack of LC_{50} data on active rats.

ANALYSIS OF DATA

COMPARISON OF PREDICTED TIMES TO EFFECT FOR EACH HAZARD.

Many species of animals have been tested at different activity levels by many investigators. An overall picture on how this complex assortment of data is related can be obtained by a direct comparison of the predicted times to effect. The goal is to find an equation or equations that best approximate the time to effect for a man engaging in light activity. From an analysis of this comparative data, the best approximations were selected to be used in the New FAA Survival Model.

The principle investigators are listed in the comparisons. It is understood that the investigators employed the following test subjects and activity levels (table 24).

TABLE 24. INVESTIGATORS, TEST SUBJECTS, AND ACTIVITY LEVELS

Investigator	Test Subjects	Activity Level	Hazards
Purser	monkeys	active	CO, CO_2
Purser	monkeys	seated	HCN
Crane	rats	motor-driven wheel	CO, HCN, acrolein, HCl
Sakurai	mice	motor-driven wheel	CO, HCN, HCl, NO_2, O_2, CO_2
Kaplan	baboons, rats	escape task	CO, HCl, acrolein
Hartzell	rats	restrained	CO, HCN, HCl
Levin	rats	restrained	CO, HCN, NO_2
Stewart	humans	light work	CO
Luft	humans	resting (at altitude)	O_2
Gray	rats	unrestrained	NO_2
Blockley	humans	resting	CO_2
Bitron & Aharonson	mice	unrestrained	SO_2
Purser*	humans	resting	CO_2, heat, O_2
Crane*	humans	resting	heat
Speitel*	rat	restrained	NO_2, acrolein
Speitel*	humans	resting	O_2, CO_2

*The investigator reviewed the literature and compiled data obtained by the works of others.

The author provided a regression equation and/or reasonable limits when not provided by the investigator.

A comparison of the predicted times to effect are tabulated (tables 25 to 33) and plotted (figures 45 to 52) for each hazard. The predictions outside the calibration range (in the tables) are indicated by light shading. Predictions outside the calibration range (in figures) are indicated by dashed lines. Lethality and incapacitation predictions are indicated by thick and thin lines respectively. The maximum concentrations observed in full-scale aircraft cabin fire tests conducted at the FAA Technical Center are also noted for each gas. The predictive equations selected for the survival model are indicated for each gas. Predictions outside this calibration range are not as reliable.

CARBON MONOXIDE. The regression equations for CO exposures are:

Purser: (Speitel) $t_i = \frac{26{,}912}{C_{co}}$ where average C•t = 26,912 ppm•min (6)

Stewart: (Purser) $t_i = \frac{36{,}177}{(C_{co})^{1.036}}$ (*light work*) when C_{CO} is expressed in ppm (2)

Crane: $t_i = \frac{25{,}017}{C_{co} - 225} + 0.3$ when $C_{CO} > 225$ ppm (5)

Sakurai: $t_i = \frac{23{,}400}{C_{co} - 160}$ when $C_{CO} > 160$ ppm (7)

Kaplan: $t_i = \frac{34{,}250}{C_{co}}$ when $C_{CO} > 95$ ppm (3)

Hartzell: $$t_i = \frac{36{,}509}{C_{CO} - 233}$$ when $C_{CO} > 233$ ppm (4)

$= \infty$ when $C_{CO} \leq 233$ ppm

Levin: (Speitel) $$t_{exposure} = \exp\,[5.85 - (3.70 \times 10^{-4})\, C_{CO}]$$ when $2000 \leq C_{CO} \leq 9000$ ppm (8)

$= [58{,}000/(C_{CO} - 4000)] + 0.4$ when $C_{CO} > 9000$ ppm

$= \infty$ when $C_{CO} < 2000$ ppm

The comparisons of predicted time to effect can be found in table 25 and figure 45.

TABLE 25. COMPARISON OF PREDICTED CARBON MONOXIDE TIMES TO EFFECT

ppm CO	t_i (min)						$t_{exposure}$ (min)
	Purser (Speitel)	Stewart (Purser)	Crane	Sakurai	Kaplan X	Hartzell	Levin (Speitel) X
100*	269.0	307.0	∞	∞	343.0	∞	∞
1000	26.9	28.2	32.5	27.9	34.3	47.6	∞
1500	17.9	18.5	19.9	17.4	22.8	28.8	∞
2000	13.5	13.8	14.4	12.7	17.1	20.7	166.0
4000	6.7	6.7	6.9	6.1	8.6	9.7	79.0
6000	4.5	4.4	4.6	4.0	5.7	6.3	37.7
6850	3.9	3.8	4.1	3.5	5.0	5.5	27.3
8000	3.4	3.3	3.5	3.0	4.3	4.7	18.0
10,000	2.7	2.6	2.9	2.4	3.4	3.7	10.1
15,000	1.8	1.7	2.0	1.6	2.3	2.5	5.7
20,000	1.3	1.3	1.6	1.2	1.7	1.8	4.0
25,000**	1.1	1.0	1.3	0.94	1.4	1.5	3.25
50,000	0.54	0.49	0.80	0.47	0.69	0.73	1.7

X Selected for the survival model.

* Note: Henderson & Haggard 9-hour (540 min) exposure of men to 100 ppm produces headache and nausea.

** Max CO concentration measured in FAA full-scale aircraft cabin fire tests > 23,000 ppm

It appears that t_i is a function of activity level: Crane's t_i's are less than Kaplan's which are less than Hartzell's. Since Kaplan's activity level was judged to be most similar to the escape task of exiting a plane and since the subjects were nonhuman primates, Kaplan's expression for t_i was selected for the FED_I model. Speitel's expression for Levin's LC_{50} data was selected for the FED_L model.

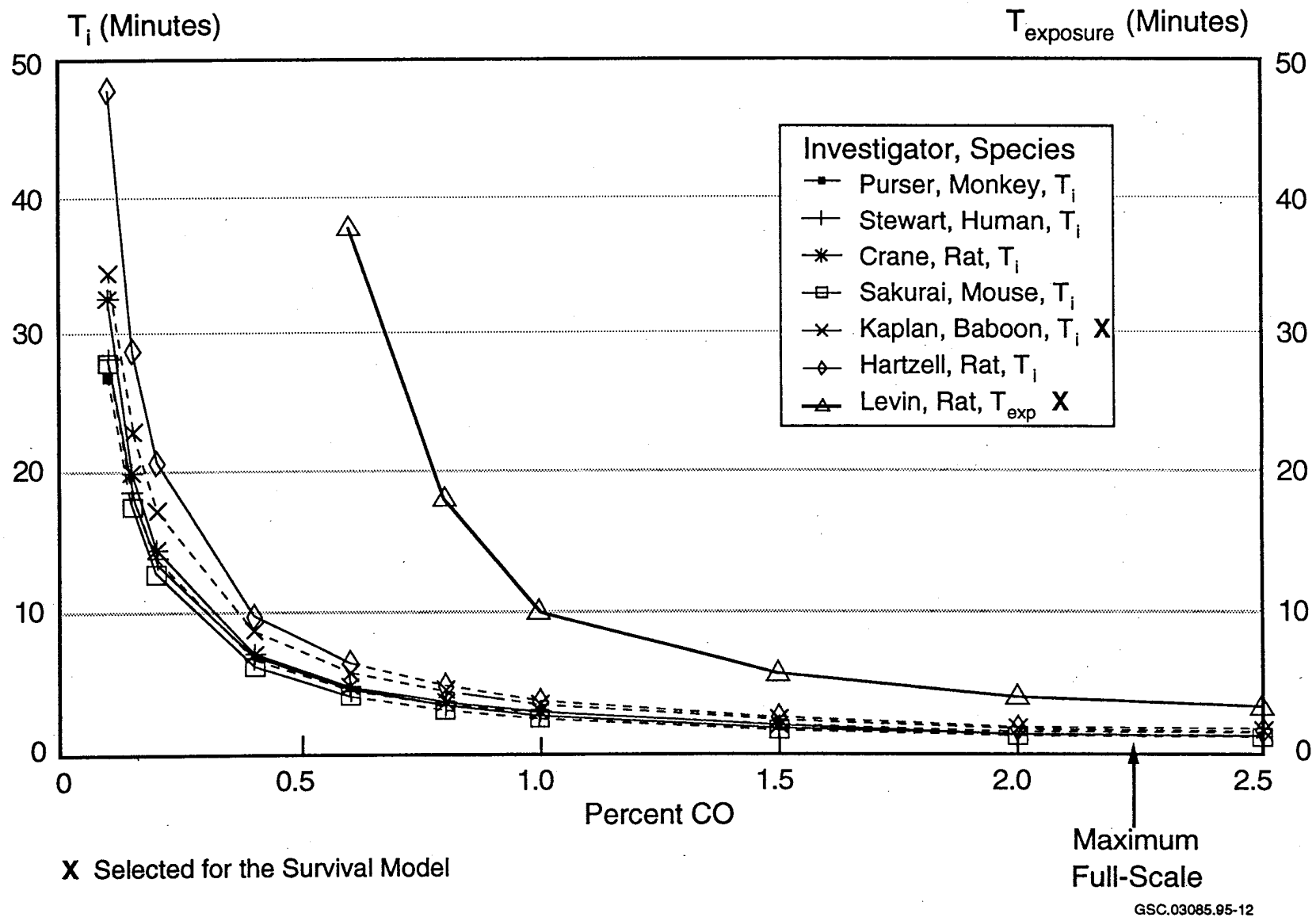


FIGURE 45. COMPARISON OF PREDICTED TIMES TO EFFECT FOR CARBON MONOXIDE

HYDROGEN CYANIDE. The regression equations for HCN exposures are:

Purser: $t_i = \frac{185 - C_{HCN}}{4.4}$ when $80 \leq C_{HCN} \leq 180$ ppm (9)

$t_i = \exp(5.396 - 0.023\ C_{HCN})$ when $C_{HCN} > 180$ ppm

Crane: $t_i = \frac{564}{C_{HCN} - 63}$ when $C_{HCN} > 63$ ppm (11)

$= \infty$ when $C_{HCN} \leq 63$ ppm

Sakurai: $t_i = \frac{491}{C_{HCN} - 25.3}$ when $C_{HCN} > 25.3$ ppm (12)

$= \infty$ when $C_{HCN} \leq 25.3$ ppm

Hartzell: $t_i = \frac{698}{C_{HCN} - 92}$ when $C_{HCN} > 92$ ppm (10)

$= \infty$ when $C_{HCN} \leq 92$ ppm

Levin: (Speitel) $t_i = \frac{2586}{C_{HCN} - 43.2}$ when $C_{HCN} > 43.2$ ppm (13)

$= \infty$ when $C_{HCN} \leq 43.2$ ppm

The comparisons of predicted time to effect can be found in table 26 and plotted in figure 46.

TABLE 26. COMPARISON OF PREDICTED HYDROGEN CYANIDE TIMES TO EFFECT

	t_i (min)				$t_{exposure}$ (min)
ppm HCN	Purser	Crane X	Sakurai	Hartzell	Levin (Speitel) X
25	36.4	∞	∞	∞	∞
75	25.0	47.0	9.9	∞	81.3
100	19.3	15.2	6.6	87.3	45.5
150	8.0	6.5	3.4	12.0	24.2
200	2.2	4.1	2.8	6.5	16.5
300**	0.22*	2.4	1.8	3.4	10.1
600	0	1.1	0.9	1.4	4.6
2400	0	0.2	0.2	0.3	1.1

X Selected for the survival model.
* Purser's equation poor fit for his data: t_i = 1.1 min at 300 ppm.
** Max HCN concentration measured in FAA full-scale aircraft fire tests = 400 ppm (post flashover).

It appears again that t_i is a function of activity level: Crane's t_i's are less than Hartzell's. Crane's activity level was judged to be the closest to the escape task of exiting a plane. Therefore, Crane's t_i expression was selected for the FED_I model. Speitel's equation for Levin's LC_{50} data was selected for the FED_L model.

HYDROGEN CHLORIDE. The regression equations for HCl exposures are:

Sakurai: $$t_i = \frac{1.03 \times 10^{+5}}{C_{HCl} - 9040} \quad \text{when } C_{HCl} > 9040 \text{ ppm} \qquad (16)$$

$$= \infty \quad \text{when } C_{HCl} \leq 9040 \text{ ppm}$$

Crane: $$t_i = 3 + \frac{3.36 \times 10^{+5}}{C_{HCl} - 300} \quad \text{when } C_{HCl} > 300 \text{ ppm} \qquad (15)$$

$$= \infty \quad \text{when } C_{HCl} \leq 300 \text{ ppm}$$

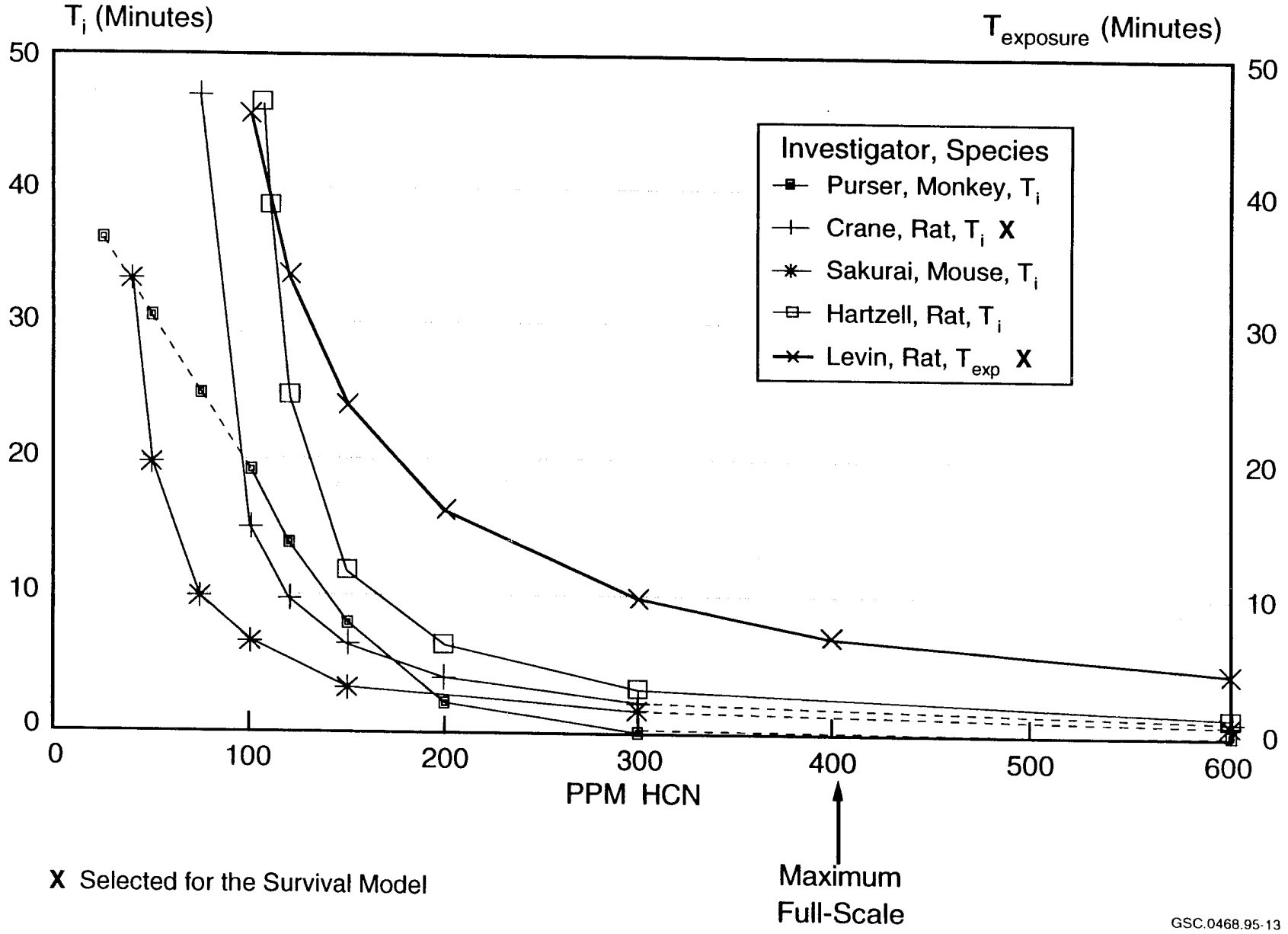


FIGURE 46. COMPARISON OF PREDICTED TIMES TO EFFECT FOR HYDROGEN CYANIDE

Corrected figure

Hartzell: $$t_{exposure} = \frac{70{,}500}{C_{HCl} - 1800} \quad \text{when } C_{HCl} > 1800 \text{ ppm} \qquad (14)$$

$$= \infty \quad \text{when } C_{HCl} \leq 1800 \text{ ppm}$$

The comparisons of predicted time to effect can be found in table 27 and figure 47.

Kaplan used a limited number of baboons for HCl 5-minute escape tests, and all animals successfully performed the escape task. His rat data is consistent with his baboon data, both for t_i and $t_{exposure}$. Crane's and Hartzell's data for 5-minute exposures matched Kaplan's fairly well. Crane's and Hartzell's expressions for t_i and $t_{exposure}$ respectively were selected for the FED_I and FED_L models as the exposure concentration range was greater and included many exposure concentrations.

TABLE 27. COMPARISON OF PREDICTED HYDROGEN CHLORIDE TIMES TO EFFECT

	t_i (min)				$t_{exposure}$ (min)		
ppm HCl	Sakurai	Crane X	Kaplan** (baboon)	(rat)	Hartzell X	Kaplan** (baboon)	(rat)
400	∞	3363.0			∞	S	N
1000	∞	486.0			∞	U	O
2000	∞	201.0			353.0	R	
3000	∞	127.0	E		58.8	V	D
4000	∞	94.0	S	E	32.0	I	A
5000*	∞	74.0	C	S	22.0	V	T
10,000	107.0	38.0	A	C	8.6	E	A
11,400	43.6	33.2	P	A	7.3		SURVIVE
14,410	19.2	26.8	E	P	5.6	NO	
15,250	16.6	25.5		E	5.2	DATA	D
16,570	13.7	20.7			4.8		E
17,290	12.5	19.8			4.6	DEATH	A
50,000	2.5	9.8			1.5		T
76,730	1.5	7.4	NO		0.9	NO	H
87,660	1.3	6.9	DATA	FAIL	0.8	DATA	
100,000	1.1	6.4		NO DATA	0.7		NO DATA

X Selected for the survival model

* Maximum concentration of HCl in full-scale aircraft cabin fire tests = 5000 ppm

** Five-minute exposure for all tests

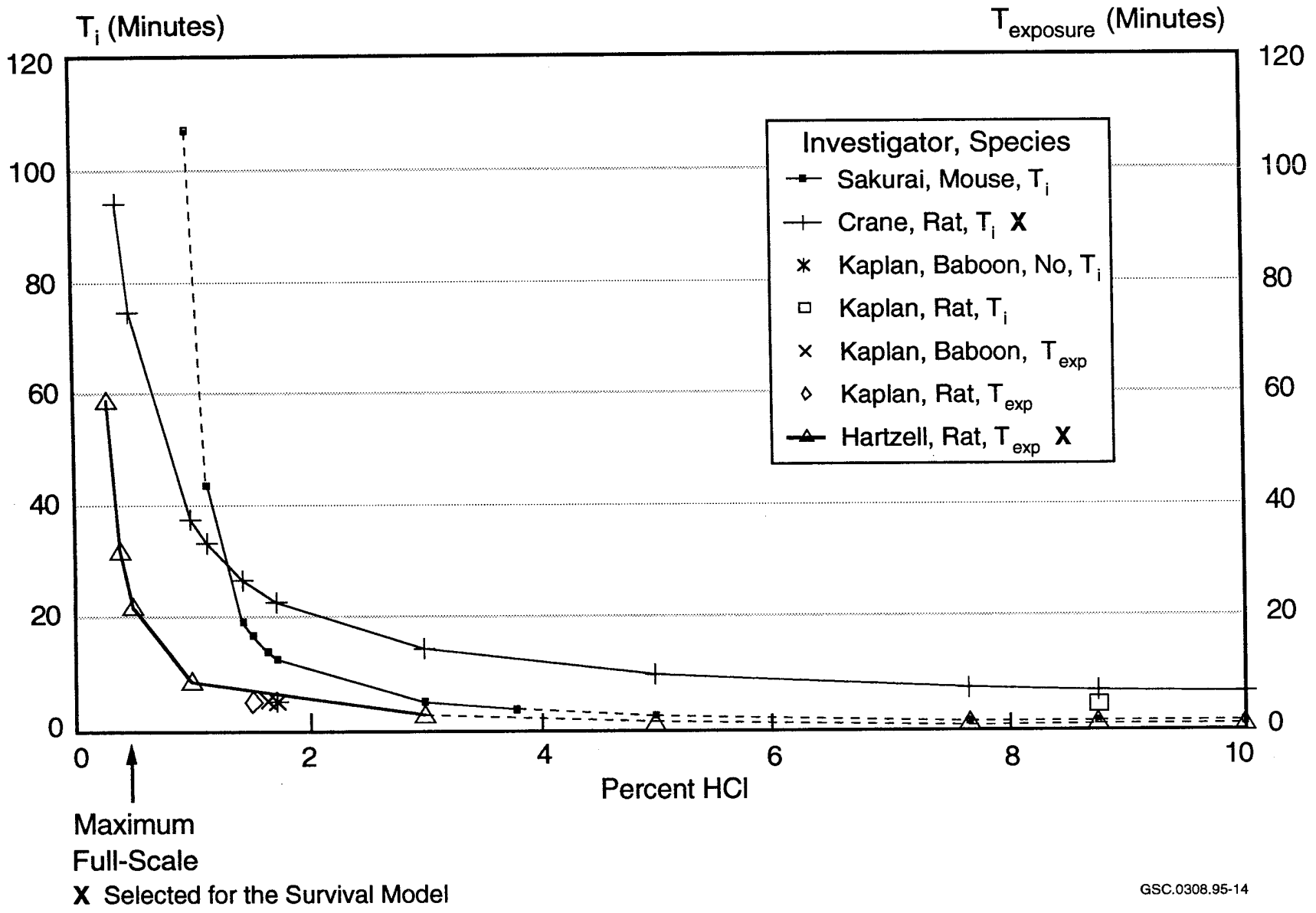


FIGURE 47. COMPARISON OF PREDICTED TIMES TO EFFECT FOR HYDROGEN CHLORIDE

ACROLEIN. The regression equations for acrolein exposures are:

Crane: $t_i = 1.5 + (4.0 \times 10^{+4}) / (C_{Acrolein} + 500)$ when $C_{acrolein} > 300$ ppm (23)

Kaplan: $t_{exposure} = \frac{3825}{C_{Acrolein}}$ (Based on estimate of 5-minute LC_{50}) (19)

Pattle & Cullumbine: $t_{exposure} = \frac{3780}{C_{Acrolein}}$ (Based on 6-hour LC_{50} of 10.5) (21)

Skogg: $t_{exposure} = \frac{4050}{C_{Acrolein}}$ (Based on 30-minute LC_{50} of 135 ppm) (20)

Pattle, Cullumbine and Skogg: (Speitel) $t_{expsoure} = \frac{3915}{C_{Acrolein}}$ (Based on average of the Ct products: (3780 + 4050)/2) when $C_{acrolein} > 5$ ppm (22)

The comparisons of predicted time to effect can be found in table 28, figure 48.

TABLE 28. COMPARISON OF PREDICTED ACROLEIN TIMES TO EFFECT

ppm Acrolein	t_i (min)	$t_{exposure}$ (min)	
	Crane X	Kaplan***	Pattle, Cullumbine & Skogg (Speitel) X
10*	∞	383.0	392.0
100**	∞	38.2	39.2
200	∞	19.1	19.6
500	41.5	7.7	7.8
1000	28.2	3.8	3.9
10,000	5.3	0.4	0.4
40,000	2.5	0.1	0.1

X Selected for the survival model.
* Iwanoff exposed cats to 10 ppm acrolein for 3.5 hours. The cats experienced a mild narcosis.
** Estimated maximum concentration of acrolein in aircraft cabin fire tests = 100 ppm.
***Based on 5-minute exposure data.

Crane's expression for t_i and Speitel's expression for $t_{exposure}$, based on the data of Pattle, Cullumbine, and Skogg were selected for the model. This $t_{exposure}$ expression is consistent with the 5-minute baboon exposure data of Kaplan.

NITROGEN DIOXIDE. The regression equations for NO_2 exposures are:

Sakurai: $$t_i = \frac{1.14 \times 10^{+4}}{C_{NO_2} - 290} \quad \text{when } C_{NO_2} > 290 \text{ ppm} \qquad (27)$$

$$t_i = \infty \quad \text{when } C_{NO_2} \leq 290 \text{ ppm}$$

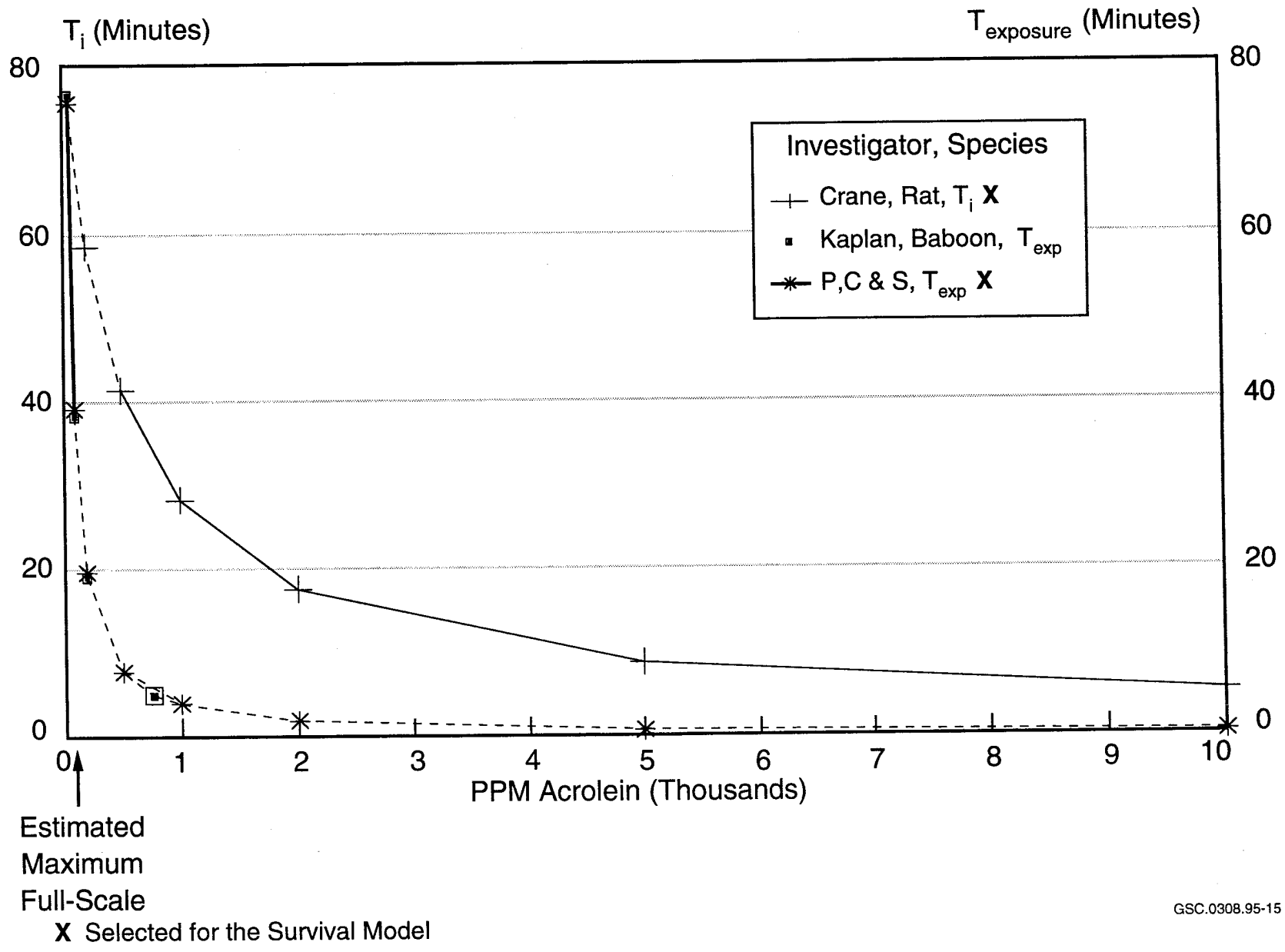


FIGURE 48. COMPARISON OF PREDICTED TIMES TO EFFECT FOR ACROLEIN

Levin, Gray, Higgins (Speitel):

$$t_{exposure} = \frac{3899}{C_{NO_2} - 71.8} \quad \text{when } C_{NO_2} > 71.8 \text{ ppm} \tag{26}$$

$$= \infty \quad \text{when } C_{NO_2} \leq 71.8 \text{ ppm}$$

Gray et al.:

$$LC_{50} = \frac{2051}{t_{exposure}^{0.609}} \tag{25}$$

The predicted times to effect can be found in table 29 and figure 49.

TABLE 29. COMPARISON OF PREDICTED NITROGEN DIOXIDE TIMES TO EFFECT

	t_i (min)	$t_{exposure}$ (min)	
ppm NO_2	Sakurai X	Levin, Gray, & Higgins (Speitel) X	Gray et al.
88	∞	240.1	175.9
200*	∞	30.4	45.7
300	1140.0	17.1	23.4
400	103.6	11.9	14.6
600	36.8	7.4	7.5
833	21.0	5.1	4.4
1000	16.1	4.2	3.3
2000	6.7	2.0	1.0
3000	4.2	1.3	0.5
4000	3.1	1.0	0.3

X Selected for survival model.
* Estimated maximum NO_2 concentration in aircraft cabin fires = 200 ppm

Sakurai's and Speitel's expressions for t_i and $t_{exposure}$ respectively were selected for the model.

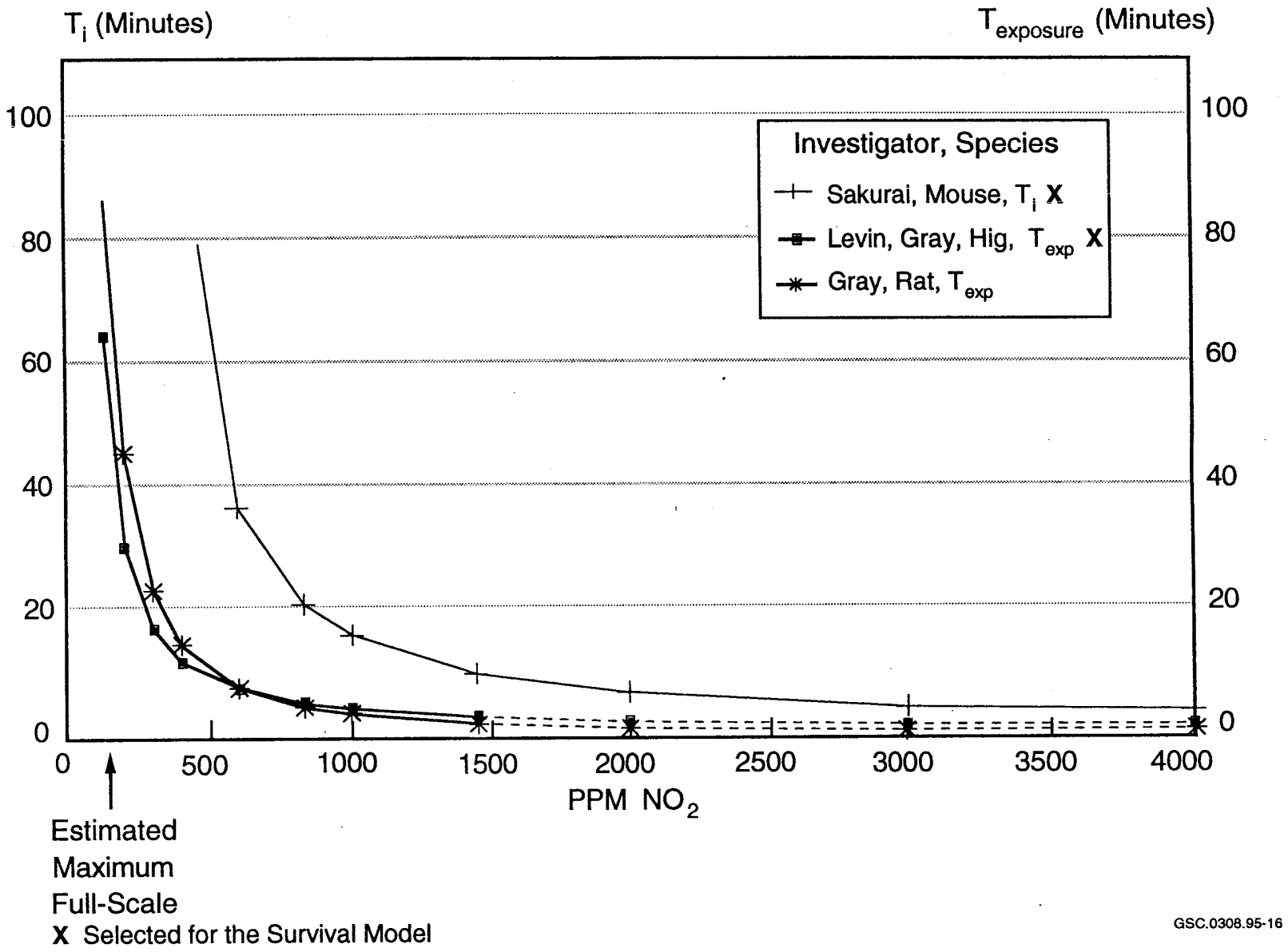


FIGURE 49. COMPARISON OF PREDICTED TIMES TO EFFECT FOR NITROGEN DIOXIDE

SULFUR DIOXIDE. The regression equation for SO_2 exposure is:

Bitron & Aharonson
(Speitel):

$$t_{exposure} = 0.633 \exp [8.18012 - (2.89037 \times 10^{-3}) C_{SO_2}] \quad \text{when } C_{SO_2} > 300 \text{ ppm} \qquad (29)$$

$$= \infty \quad \text{when } C_{SO_2} \leq 300 \text{ ppm}$$

The predicted time to effect can be found in table 30.

TABLE 30. PREDICTED SULFUR DIOXIDE TIMES TO EFFECT

ppm SO_2	$t_{exposure}$ (min) Bitron & Aharonson (Speitel) X
200	∞
300*	950.0
600	398.6
900	167.6
1200	70.4
1500	29.5
1800	12.4
2300	2.9
5000	0.0

X Selected for survival model.
* Estimated maximum SO_2 concentration in aircraft cabin fires = 390 ppm

Speitel's expression for $t_{exposure}$ was selected for the model.

LOW-OXYGEN HYPOXIA. The regression equations for low-oxygen exposures are:

Sakurai: $$t_i = \frac{16.6}{9.77 - C_{Oxygen}} \quad \text{when } C_{Oxygen} < 9.77\% \qquad (38)$$

$$= \infty \quad \text{when } C_{Oxygen} \geq 9.77\%$$

Luft (Purser): $$t_i = \exp\,[8.13 - 0.54\,(20.9 - C_{Oxygen})] \quad \text{when } C_{Oxygen} < 20.9\% \qquad (35)$$

Luft (Speitel): $$t_i = \exp\,[8.55 - 0.511\,(20.9 - C_{Oxygen})] \quad \text{when } C_{Oxygen} < 11.0\% \qquad (37)$$

$$= \infty \quad \text{when } C_{Oxygen} \geq 11.0\%$$

The comparisons of predicted time to effect can be found in table 31 and are plotted in figure 50.

TABLE 31. COMPARISON OF PREDICTED LOW-OXYGEN HYPOXIA TIMES TO EFFECT

% O_2	t_i (min)		
	Sakurai (mouse)	Luft (Purser) (man)	Luft (Speitel) (man) X
0	1.7	0.0	0.1
2*	2.1	0.1	0.3
4	2.9	0.4	0.9
6	4.4	1.1	2.5
7	6.0	1.9	4.3
8	9.4	3.2	7.1
9	21.6	5.5	11.8
10**	∞	9.4	19.7
11	∞	16.2	∞
12	∞	27.7	∞
13	∞	47.6	∞

X Selected for survival model.

* Minimum O_2 concentration measured in full-scale aircraft cabin fire tests = 3% (post flashover)

** Purser - 30-minute exposure to 10% O_2 (4 seated monkeys) - conscious, muscle weakness & impaired reflexes, Venous P_{O_2} = 10 mm Hg.

** Luria & Knight - 15-minute maximum exposure to 10% O_2 (6 humans) - dizziness, lightheadedness, air hunger. (see text)

Speitel's expression for t_i was based on the same data as Purser's (men at altitude), but Speitel included additional corrections for sea level. Speitel's predicted t_i's for 10% oxygen are consistent with Purser's normobaric 10% oxygen monkey data, Luria & Knight's normobaric human exposure data, and Slobodnik's normobaric 6%, 7%, and 8% oxygen human data. Speitel's expression was selected for the FED_I model. Note that these data were based on men at rest. Physical exertion greatly increases the severity of the exposure and will lead to earlier incapacitation[59].

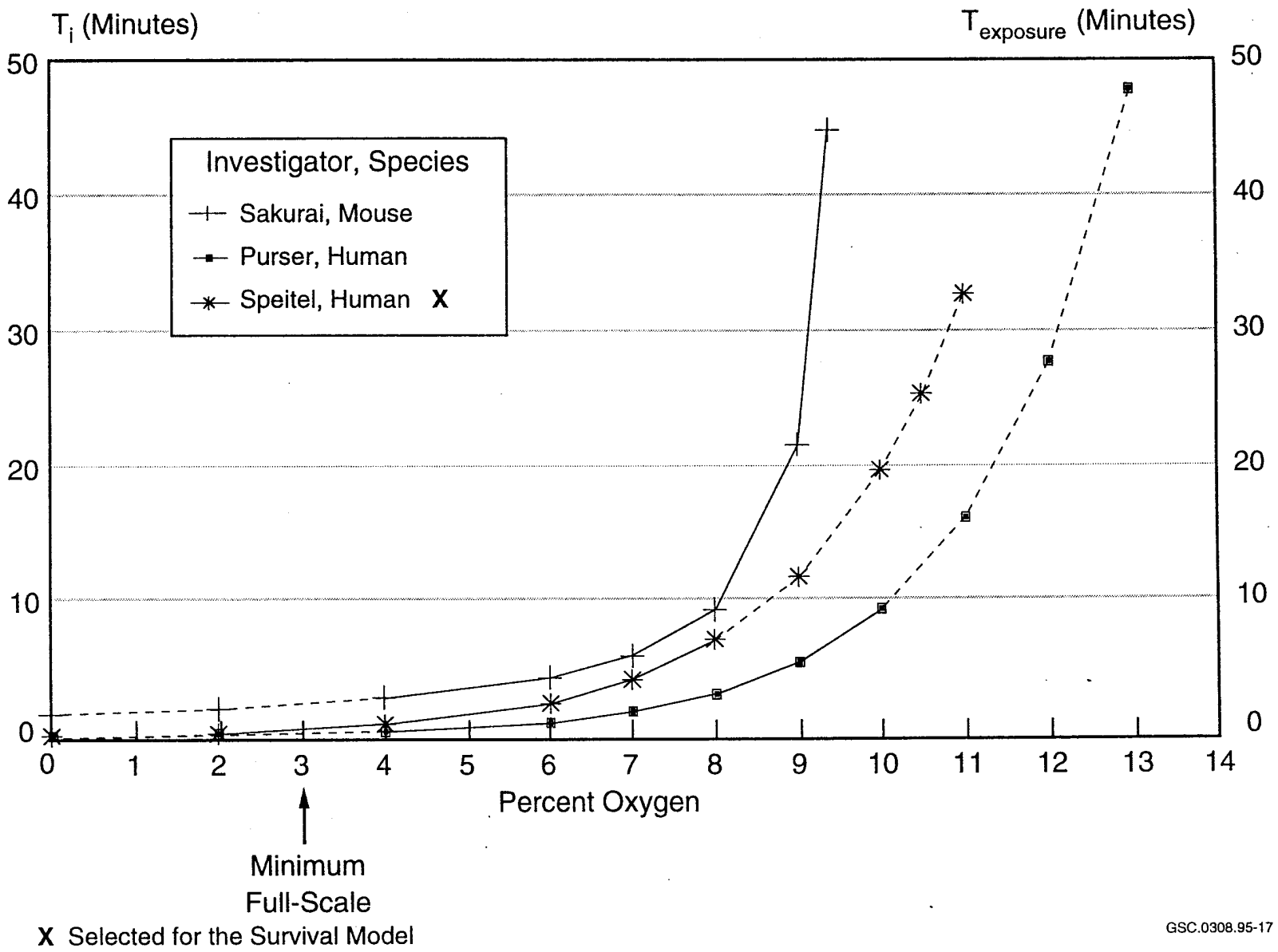


FIGURE 50. COMPARISON OF PREDICTED TIMES TO INCAPACITATION FOR LOW OXYGEN

CONVECTIVE HEAT. The regression equations for collapse due to exposure to convective heat are:

Crane: $t_c = \frac{4.1 \times 10^{+8}}{T^{3.61}}$ where T = °C (39)
Assume $t_c = \infty$ when T < 50°C

Purser: $t_c = \exp(5.1849 - 0.0273 \times T)$ where T = °C (40)

Blockley: t_c *from temp. vs. time curve*

The comparisons of predicted time to effect can be found in table 32 and figure 51.

TABLE 32. COMPARISON OF PREDICTED TIMES TO THERMAL COLLAPSE

Temperature		t_c (min)			
°C	°F	Crane X	Purser (avg. humid & dry)	Blockley's Curve Humid	Dry
50	122	302.0	45.6	70.0	13,500.0
100	212	24.7	11.6	8.5	13.5
150	302	5.7	3.0	2.3	
200	392	2.0	0.76 (46s)	0.70 (42s)	
250	482	0.90	0.19 (11s)	0.37 (22s)	
300	572	0.47 (28s)	0.05 (3s)	0.17 (10s)	
400	752	0.17 (10s)	0.00	0.05 (3s)	
500	932	0.07 (4s)	0.00	0.02 (1s)	
600	1112	0.04 (2s)	0.00	0.01	
700*	1292	0.02 (1s)	0.00	0.00	

X Selected for survival model.
* Maximum temperature measured in full-scale aircraft cabin fire tests > 700°C
Assume $t_c = \infty$ when T < 50°C

Crane's expression for t_c was based on clothed subjects and is therefore based on the most realistic exposure conditions. Crane's expression was selected for the model.

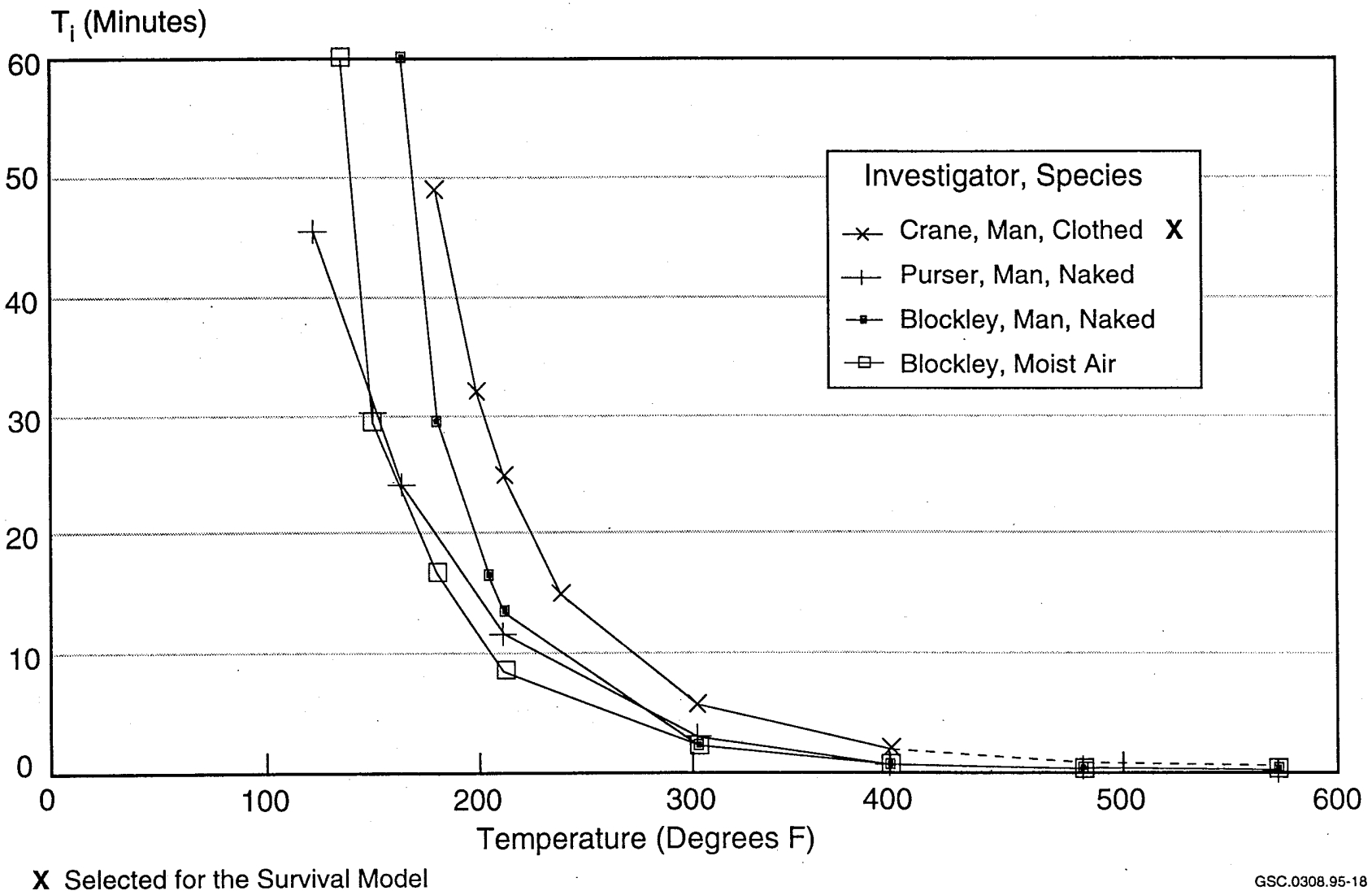


FIGURE 51. COMPARISON OF PREDICTED TIMES TO INCAPACITATION FOR HEAT

CARBON DIOXIDE. The regression equations for CO_2 exposures are:

Purser: $t_i = exp\ (6.1623 - 0.5189 \times C_{CO_2})$ where C_{CO_2} = % CO_2 (31)

Purser/ Speitel: $t_i = exp\ (6.1623 - 0.5189 \times C_{CO_2})$ when $C_{CO_2} > 7.0\%$ (31, 32)

$= 2193.8 - 311.6 \times C_{CO_2}$ when $5.5 \leq C_{CO_2} \leq 7.0\%$

$= \infty$ when $C_{CO_2} < 5.5\%$

Blockley: (Speitel) $t_i = exp\ (11.4 - 1.14 \times C_{CO_2})$ when $C_{CO_2} > 6.0\%$ CO_2 (30)

Sakurai: $t_i = \dfrac{9.97}{C_{CO_2} - 20.0}$ when $C_{CO_2} > 20\%$ (33)

Purser's expression for t_i is a good fit for the higher CO_2 concentrations. Speitel's linear expression is a reasonable fit for the lower concentrations. The assumption was made that $t_i = \infty$ for concentrations less than 5.5% CO_2, which is supported by previously discussed human exposure data. Purser's equation with Speitel's expression for low concentrations (0% - 7.0% O_2) was selected for the FED_I model.

The comparisons of predicted times to effect can be found in table 33 and figure 52.

TABLE 33. COMPARISON OF PREDICTED CARBON DIOXIDE TIMES TO EFFECT

% CO_2	t_i (min)			
	Purser	Purser/Speitel X	Blockley	Sakurai
4	59.5	∞	∞	∞
5	35.4	∞	∞	∞
6	21.1	324.0	95.6	∞
7	12.6	12.6	30.6	∞
8	7.5	7.5	9.8	∞
9	4.5	4.5	3.1	∞
10*	2.6	2.6	1.0	∞
11	1.6	1.6	0.32	∞
12	0.9	0.9	0.10	∞
18	0.04	0.04	0.00	∞
21	0.01	0.01	0.00	10.0

X Selected for survival model.
* Maximum concentration measured in FAA full-scale aircraft fire test.

EFFECT OF CO_2 ON INCREASED UPTAKE OF OTHER GASES.

An important assumption made in the model was that increased uptake of other gases matches the predicted RMV increase due to CO_2. This assumption was applied to all gases except CO_2 and O_2 on the basis of the following observations:

1. Acid gases. Inspired acid gases must travel through the moist respiratory tract. Therefore since acid gases HF, HCl, HBr, and HCN are very soluble in water, they should be collected quantitatively.

2. Carbon Monoxide:

   a. The apparent increased uptake of CO in presence of 5% CO_2 based on rat LC_{50} data for 5- to 60-minute exposures ranged from 1.2- to 1.6-fold respectively.

   b. The rate of formation (in rat) of COHb (before saturation) was 1.5 times greater for rats exposed to 2500 ppm CO in the presence of 5.25% CO_2 than for rats exposed to 2500 ppm CO without 5.25% CO_2.

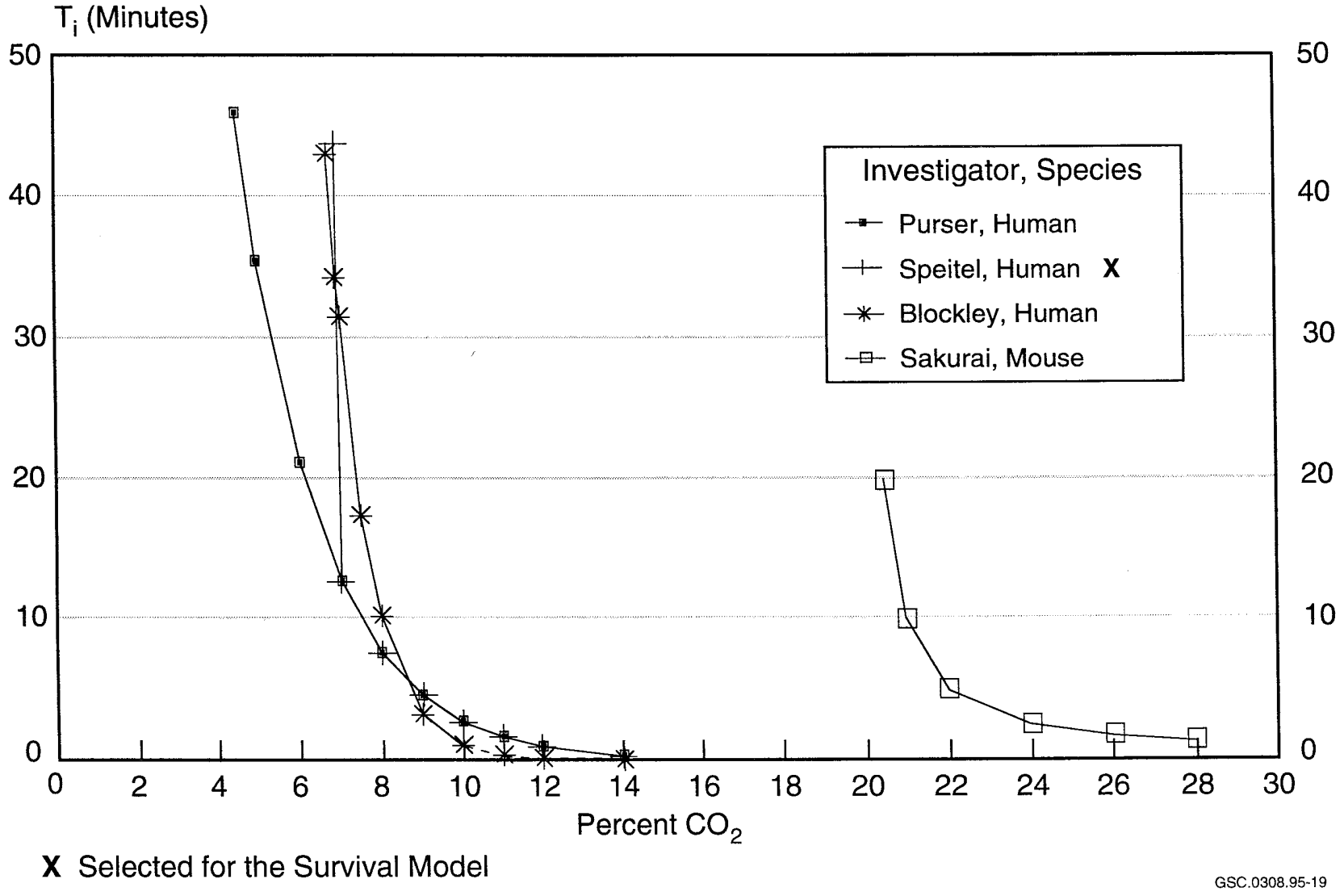


FIGURE 52. COMPARISON OF PREDICTED TIMES TO INCAPACITATION FOR CARBON DIOXIDE

c. The RMV for rats exposed to 5% CO_2 is a factor of 2.2 greater than control rats.

The uptake of CO in presence of 5% CO_2 is greater than in its absence but does not match the predicted 2.2 fold RMV increase in rats. Erring on the side of safety, it was assumed in the model that CO uptake matched the predicted RMV increase in humans.

3. Nitrogen Dioxide: The multiplication factor for the apparent enhanced uptake of NO_2 in the presence of 5% CO_2 matches the predicted RMV increase in rats (2.2 vs. 2.2).

4. Oxygen: Carbon Dioxide has been shown to have a marked beneficial effect of elevating cerebral P_{O_2} levels for low inspired levels of oxygen in monkeys. The relationship between time to effect and levels of O_2 and CO_2 is not known for a range of CO_2 concentrations. Erring on the side of safety, the beneficial effect of CO_2 on low O_2 was not considered in the new survival model.

Note that the increase in RMV due to the presence of CO_2 (V_{CO_2}) appears to be species dependent. In the presence of 5% CO_2 the V_{CO_2}'s were found to be 2.2 for the rat, 2.2 for the guinea pig, 2 for the hamster, 3.45 for man, and 3 for the monkey. In the presence of 10% CO_2 V_{CO_2}'s were found to be 3 for the hamster, 5.5 for the anesthetized baboon, and 12.0 for man.

DEVELOPMENT OF NEW FAA SURVIVAL MODEL

PREVIOUS FAA SURVIVAL MODEL.

The previous FAA survival model[81,82] computes incapacitation in a fire environment composed of a number of toxic gases at elevated temperatures, each varying with time. The major assumptions were twofold: (1) The hazards are additive, and (2) the $C_i t_i$ product is constant. $C_i t_i = K_i$

The Fractional Effective Dose (FED) is used in estimating the toxic hazard of a hot gas mixture as a function of exposure time for constantly changing gas concentrations and temperatures.

The Fractional Effective Dose (for a constant concentration of the toxicant) is the dose received up to time t of the exposure divided by the effective Ct dose to cause an effect (e.g., incapacitation, death).

$$FED_i = \frac{Dose\ Recevied\ at\ Time\ t}{Effective\ C \cdot t\ Dose\ to\ Incapacitation} \tag{45}$$

$$= \frac{C_i t}{K_i}\ For\ a\ constant\ concentration\ of\ gas\ i \tag{46}$$

$$FED_i = \int \frac{C_i\, dt}{K_i}\ For\ a\ changing\ concentration\ of\ gas\ i \tag{47}$$

where:

C_i = concentration of gas species i
t_i = time of incapacitation
K_i = incapacitative dose of gas species i, a constant
FED_i = fractional effective dose, or the ratio of the actual dose of gas species i to the incapacitation dose

The incapacitation dose constants (K_i) were calculated from either Threshold Limit Values (TLV) or the best available data in the literature (when the model was developed in 1982) and are tabulated in table 34 along with estimated 5-minute hazard limits.

The TLVs are time-weighted average concentrations for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed day after day without adverse effect. The basis on which the values are established differ from gas to gas; protection against impairment of health may be a guiding factor for some, whereas reasonable freedom from irritation, narcosis, nuisance, or other forms of stress may form the basis for others.

To calculate K_i from TLVs:

$$K_i = C_i t_i = 480 \cdot TLV$$ where: 480 = minutes in 8-hour workday
TLV based on 8-hour day (in ppm)

TABLE 34. PREVIOUS FAA SURVIVAL MODEL WITH 5-MINUTE HAZARD LIMIT

Gas Species i	TLV (ppm)	K_i (ppm • min)	5-minute Hazard Limit (ppm)
CO	50.0	24,000	4800
CO_2	5000.0	2,400,000 750,000[a]	480,000 150,000[a]
HCN	1.0	480	96
HF	3.0	1440	288
HCl	5.0	2400	480
HBr	3.0	1440	288
NO_2	5.0	2400	480
SO_2	5.0	2400	480

where a = Adjusted. Literature surveyed to find data closest to 5-minute survival time.

The effect of elevated temperature on incapacitation was taken into account by utilizing the empirically based curve fit derived by Crane, shown below.

$$t_c = Q_o / T^{3.61}$$ (39)

where t_c = time to thermal collapse (incapacitation) in minutes
T = air temperature °C
$Q_o = 4.1 \times 10^{+8}$

The Thermal Fractional Effective Dose, FED_T, becomes:

$$FED_T = \int \frac{dt}{t_c} = \int \frac{T^{3.61} dt}{4.1 \ x \ 10^{+8}} \quad \text{when T = °C} \tag{48}$$

Therefore, assuming the hazards to be additive, the fractional effective dose becomes:

$$FED = FED_T + \Sigma \ FED_i = \int \frac{T^{3.61} \ dt}{4.1 \ x \ 10^{+8}} + \Sigma \int \frac{C_i \ dt}{K_i} \tag{49}$$

The hypothetical time of incapacitation for all hazards is the time at which FED = 1.0 .

NEW FAA SURVIVAL MODEL.

For most toxic fire gases, incapacitation occurs when the victim has inhaled a particular Ct product dose.

The fractional effective dose (FED) is useful in estimating the toxic hazard of a substance at a particular point in time. The fractional effective dose (for a constant concentration of the toxicant) is the dose received up to time t into the exposure divided by the effective Ct dose to cause an effect (e.g., incapacitation, death). For a constant concentration of toxicant:

$$FED_{effect} = \frac{dose \ received \ at \ time \ t}{effective \ Ct \ dose \ to \ cause \ effect} = \frac{Ct}{Ct_{effect}} = \frac{t}{t_{effect}} \tag{50}$$

Incapacitation is predicted to occur when FED_I is equal to one.

For substances obeying Haber's Rule, the denominator (Ct_i) of the equation is a constant. For substances deviating from Haber's Rule, the denominator for each time segment during the fire is the Ct product dose at which incapacitation would occur at the concentration during that time segment.

If the concentration varies with time, the fractional doses per minute for a pure gas can be summed for each minute of a j-minute exposure, assuming concentrations are constant within each minute:

$$FED_I = \Sigma\ (1/t_{i1} + 1/t_{i2} + \ldots\ 1/t_{ij}) \tag{51}$$

Or more generally:

$$FED_I = \int_o^t \frac{dt}{t_i} \qquad \text{where } t_i \text{ is a function of gas concentration.} \tag{52}$$

Likewise, lethality is predicted to occur for 50% of the exposed subjects when FED_L is equal to 1. For a varying concentration with time:

$$FED_L = \int_o^t \frac{dt}{t_{exposure}} \tag{53}$$

where $t_{exposure}$ is a function of gas concentration, $t_{exposure}$ is an expression for the exposure time at which LC_{50} occurs for a given concentration.

The FED_I can be calculated for a mixture of n gases and heat. For any mixture of n gases, assuming the hazards of each gas are additive:

$$FED_I = FED_{I\ Gases} + FED_{I\ Heat} \tag{54}$$

$$FED_I = \sum_{k=1}^{n} \int \frac{dt}{t_{ik}} + \int \frac{dt}{t_{i\ Heat}} \tag{55}$$

$$FED_I = \int \frac{dt}{t_{i1}} + \int \frac{dt}{t_{i2}} + \ldots \int \frac{dt}{t_{in}} + \int \frac{dt}{t_{i\ Heat}} \tag{56}$$

Two separate models are used independently to determine the combined hazard. The FED_I model uses t_i data for gases and heat to determine the ability to escape from a burning aircraft cabin. The FED_L model uses LC_{50} animal data to determine the ability to survive postexposure.

Regression equations for t_i and $t_{exposure}$ were selected with a preference for active animals with a level of activity that best approximated an escape task. All regression equations for t_i were based on active animals with the exception of CO_2, O_2, and heat. All regression equations for $t_{exposure}$ were based on inactive animals with the exception of acrolein.

Equations based on primate data were selected wherever possible. Crane's regression equation for $t_{i\ Heat}$ (t_c) was selected as it represented a more realistic exposure. It was based on data using clothed subjects as opposed to nude subjects.

The multiplication factor for the enhanced uptake of other gases, V_{CO_2}, was factored into the concentration term in the regression equations for all hazards with the exception of CO_2, O_2, and temperature,

where

$$V_{CO_2} = \frac{exp\ (0.2496\ x\ C_{CO_2} + 1.9086)}{6.8} \qquad \text{where } C_{CO_2} = \%\ CO_2 \tag{42}$$

These FEDs can be calculated if the various gas concentrations and cabin temperatures are known as a function of time, obtained by instrumented measurements in experimental aircraft cabin fires.

Time is expressed in minutes and temperature in degrees celsius for the FED_I and FED_L equations listed below.

A basic program which calculates and plots FED_I as a function of time can be found in appendix C.

$$FED_I = FED_{I\ Gases} + FED_{I\ Heat} \tag{54}$$

$$= FED_{I\ CO} + FED_{I\ HCN} + FED_{I\ HCl} + FED_{I\ HF} + FED_{I\ HBr} + FED_{I\ Acrolein} + FED_{I\ CO_2} + FED_{i\ NO_2} + FED_{I\ Low\ O_2} + FED_{I\ Heat} \tag{57}$$

$$= \frac{1}{3.4250} \int (V_{CO_2}\ x\ C_{CO})\ dt \qquad \text{when } V_{CO_2} \text{ x } C_{CO} > 0.01\% \tag{58}$$

$$+ \int \frac{(V_{CO_2}\ x\ C_{HCN} - 63)\ dt}{564} \qquad \text{when } V_{CO_2} \text{ x } C_{HCN} > 63 \text{ ppm}$$

$$+ \int \frac{dt}{3 + [(3.36 \times 10^{+5}) / (V_{CO_2} \times C_{HCl} - 300)]}$$ when V_{CO_2} x C_{HCl} > 300 ppm

$$+ \int \frac{dt}{3.0 + [(1.53 \times 10^{+5}) / (V_{CO_2} \times C_{HF} - 136)]}$$ when V_{CO_2} x C_{HF} > 136 ppm

$$+ \int \frac{dt}{3 + [(3.36 \times 10^{+5}) / (V_{CO_2} \times C_{HBr} - 300)]}$$ when V_{CO_2} x C_{HBr} > 300 ppm

$$+ \int \frac{dt}{1.50 + [(4.0 \times 10^{+4}) / (V_{CO_2} \times C_{Acrolein} + 500)]}$$ when V_{CO_2} x $C_{Acrolein}$ > 300 ppm

$$+ \int \frac{(V_{CO_2} \times C_{NO_2} - 290)\, dt}{1.14 \times 10^{+4}}$$ when V_{CO_2} x C_{NO_2} > 290 ppm

$$+ \int \frac{dt}{exp\ (6.1623 - 0.5189 \times C_{CO_2})}$$ when C_{CO_2} > 7.0%

$$+ \int \frac{dt}{2193.8 - 311.6 \times C_{CO_2}}$$ when $5.5 \leq C_{CO_2} \leq 7.0\%$

$$+ \int \frac{dt}{exp\ (8.55 - 0.511\ (20.9 - \%\ O_2)}$$ when % O_2 < 11%

$$+ \frac{1}{4.1 \times 10^{+8}} \int T^{3.61} dt$$ when T > 50°C

Note the FED_I equation for low O_2 is based on data for men at rest. The FED_I for men escaping from an aircraft fire would probably be higher than predicted by the above equation.

$$FED_L = FED_{L\ Gases} + FED_{L\ Heat} \tag{59}$$

$$\begin{aligned} FED_L = {} & FED_{L\ CO} + FED_{L\ HCN} + FED_{L\ HCl} + FED_{L\ HF} + FED_{L\ HBr} \\ & + FED_{L\ Acrolein} + FED_{L\ SO_2} + FED_{L\ CO_2} + FED_{L\ NO_2} \\ & + FED_{L\ HCHO} + FED_{L\ LOW\ O_2} + FED_{L\ Heat} \end{aligned} \tag{60}$$

$$FED_L = \int \frac{dt}{[58{,}000/(V_{CO_2} \times C_{CO} - 4000)\ ] + 0.4} \quad \text{when } V_{CO_2} \times C_{CO} > 9000 \text{ ppm} \tag{61}$$

$$+ \int \frac{dt}{exp\ [5.85 - (3.70 \times 10^{-4})\ (V_{CO_2} \times C_{CO})]} \quad \text{when } 2000 \text{ ppm} \le V_{CO_2} \times C_{CO} \le 9000 \text{ ppm}$$

$$+ \frac{1}{2586} \int [(V_{CO_2} \times C_{HCN}) - 43.2]\ dt \quad \text{when } V_{CO_2} \times C_{HCN} \ge 43.2 \text{ ppm}$$

$$+ \int \frac{(V_{CO_2} \times C_{HCl} - 1800)\ dt}{70{,}500} \quad \text{when } V_{CO_2} \times C_{HCl} \ge 1800 \text{ ppm}$$

$$+ \int \frac{(V_{CO_2} \times C_{HF} - 818)\ dt}{32{,}045} \quad \text{when } V_{CO_2} \times C_{HF} \ge 818 \text{ ppm}$$

$$+ \int \frac{(V_{CO_2} \times C_{HBr} - 1800)\ dt}{70{,}500} \quad \text{when } V_{CO_2} \times C_{HBr} \ge 1800 \text{ ppm}$$

$$+ \frac{1}{3825} \int (V_{CO_2} \; x \; C_{Acrolein}) \; dt \qquad \text{when } C_{acrolein} > 5 \text{ ppm}$$

$$+ \int \frac{dt}{0.633 \exp\left[8.18012 - (2.89037 \; x \; 10^{-3}) \; (V_{CO_2} \; x \; C_{SO_2})\right]} \qquad \text{when } V_{CO_2} \text{ x } C_{SO_2} \geq 300 \text{ ppm}$$

$$+ \int \frac{(V_{CO_2} \; x \; C_{NO_2} - 71.8) \; dt}{3899} \qquad \text{when } V_{CO_2} \text{ x } C_{NO_2} > 71.8 \text{ ppm}$$

$$+ \frac{1}{32{,}000} \int (V_{CO_2} \; x \; C_{HCHO}) \; dt$$

$$+ FED_{L\,CO_2} \qquad \text{(unknown)}$$

$$+ FED_{L\,Low\,O_2} \qquad \text{(unknown)}$$

$$+ FED_{L\,Heat} \qquad \text{(unknown)}$$

Note the FED_L values for all gases given in the above formula, with the exception of acrolein, were based on data for restrained rats. The FED_L for an individual escaping from an aircraft fire would probably be higher for these gases.

A high approximation for the missing fractional lethal effective doses can be obtained by substituting the FED_I for CO_2, Low O_2, and heat. Since much higher CO_2 concentrations are needed to kill than to incapacitate, this approximation results in a large overestimation of FED_L.

COMPARISON OF OLD AND NEW MODELS.

A comparison of the old and new model $C \bullet t_i$ products can be found in table 35.

TABLE 35. COMPARISON OF OLD AND NEW SURVIVAL MODEL C•t PRODUCTS

Gas	Conc. Range of Exposure	New Model	Old Model
		$C \bullet t_i$	K_i
CO	0.1% - 5.0%	3.425%•min	2.400%•min
HCN	75-273 ppm	1642-764 ppm•min	480 ppm•min
HCl	2300-94,000 ppm	422,400-516,800 ppm•min	2400 ppm•min
Acrolein	580-41,550 ppm	22,330-101,849 ppm•min	48 ppm•min
CO_2	5.51% - 12%	2,628%-0.76%•min	75%•min (a)
NO_2	300-4000 ppm	342,000-12,400 ppm•min	2400 ppm•min

(a) Adjusted. Literature surveyed to find data closest to 5-minute survival time

The new model predicts that HCl, acrolein, and NO_2 are far less of an incapacitative hazard than predicted in the old model. HCN, CO_2, acrolein, and NO_2 have a broad range of C•t products in the new model. HCN, CO_2, acrolein, and NO_2 deviate markedly from Haber's Rule. Comparison plots for predicted t_i's by the old and new model for a range of concentrations for HCN and CO_2 can be found in figures 53 and 54.

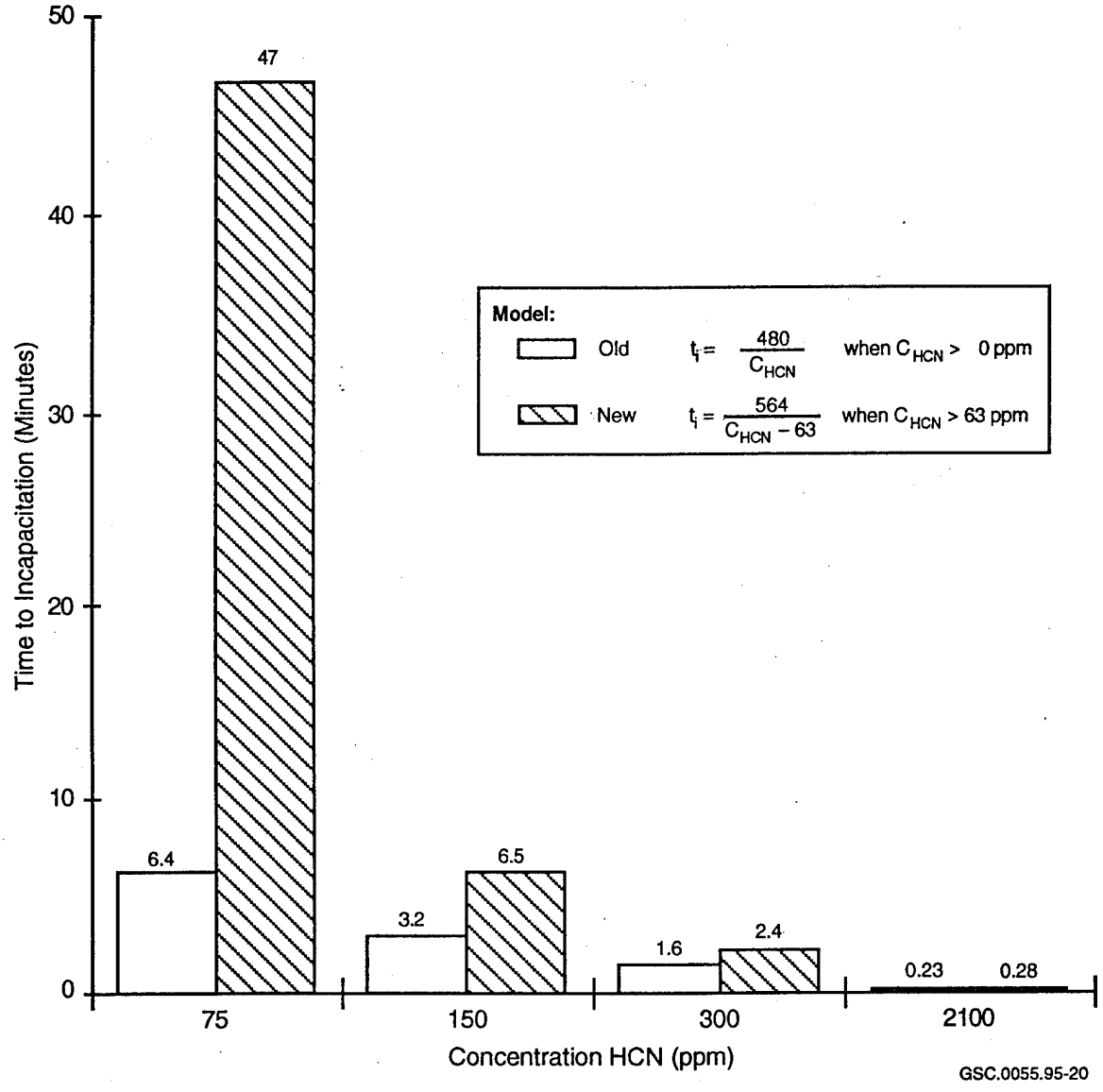


FIGURE 53. COMPARISON OF OLD AND NEW SURVIVAL MODELS FOR HYDROGEN CYANIDE AT LOW, MEDIUM, AND HIGH FULL-SCALE CONCENTRATIONS

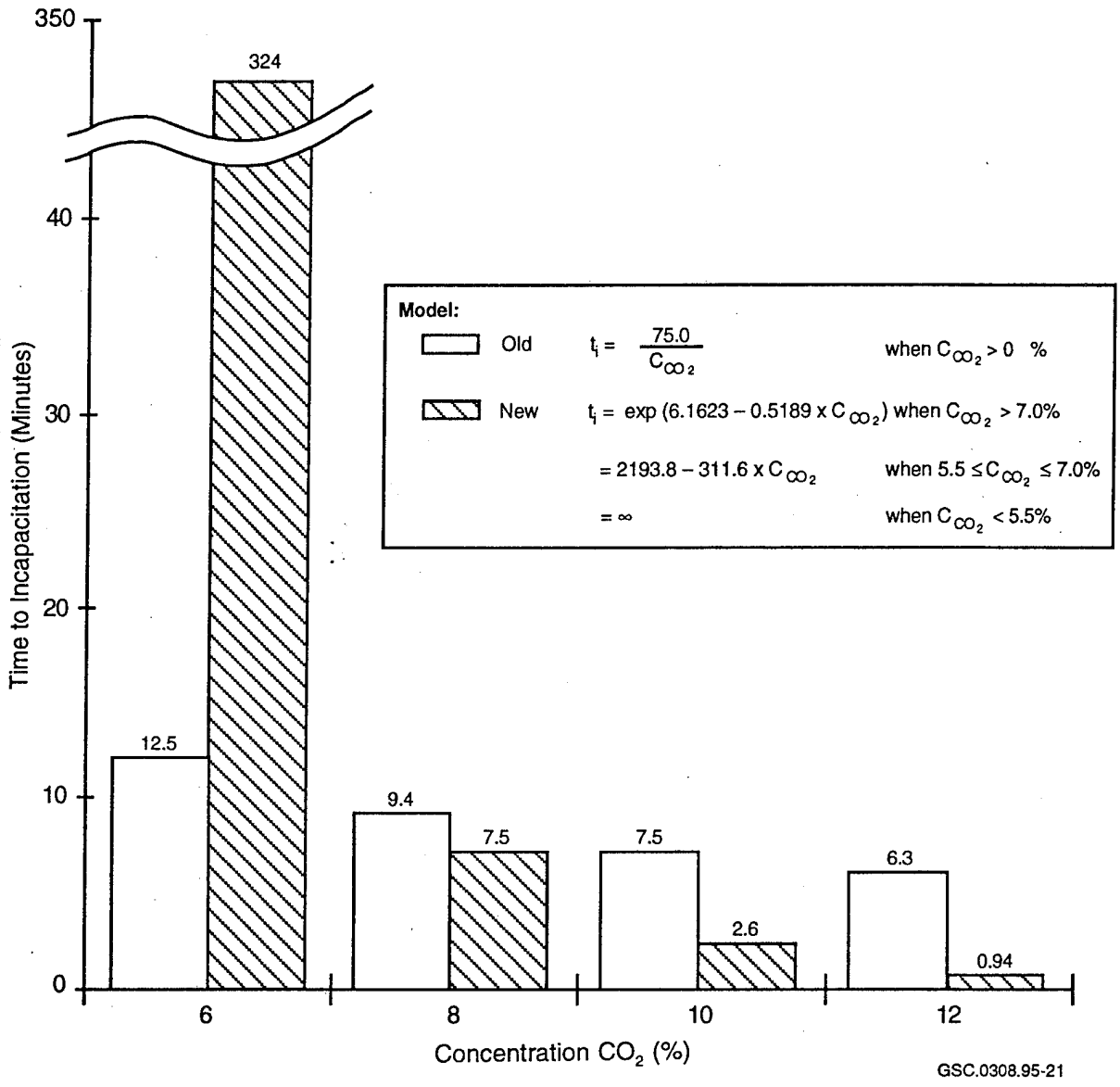


FIGURE 54. COMPARISON OF OLD AND NEW SURVIVAL MODELS FOR CARBON DIOXIDE AT LOW, MEDIUM, AND HIGH FULL-SCALE CONCENTRATIONS

The concentrations predicted to give a 5-minute time to effect are listed for the old and new models in table 36.

TABLE 36. PREDICTED CONCENTRATIONS TO GIVE A 5-MINUTE TIME TO EFFECT

		Incapacitation Concentration			Lethality Concentration (LC_{50})	
Gas	Units	Old Model	New Model	New Model with 5% CO_2	New Model	New Model with 5% CO_2
CO	ppm	4800	6850	1940	16,600	4810
HCN*	ppm	96	176	51.0	560	162
HCl*	ppm	480	168,300	48,783	15,900	4609
HF*	ppm	288	76,636	22,213	7227	2095
HBr*	ppm	288	168,300	48,783	15,900	4609
Acrolein*	ppm	9.6	10,928	3168	783	227
CO_2	%	15 (a)	8.8	N/A	ND	ND
O_2	%	N/A	7.2	7.2	ND	ND
NO_2*	ppm	480	2570	745	852	247
SO_2*	ppm	488	ND	ND	2115	613
Temp	°C	156	156	156	ND	ND

ND = Not Determined
* = Causes postexposure lethality
a = Adjusted. Literature surveyed to find data closest to 5-minute survival time

APPLICATION OF NEW MODELS TO FULL-SCALE FIRE DATA

PREDICTION OF TIME TO EFFECT FOR FULL-SCALE FIRE TESTS.

The maximum gas concentrations measured or estimated in the full-scale cabin fire tests at the FAA Technical Center are listed in table 37 along with the predicted times to effect for each gas obtained using the new FAA Survival Model.

TABLE 37. PREDICTED TIMES TO EFFECT FOR MAXIMUM FULL-SCALE FIRE GAS CONCENTRATIONS

Gas	Maximum Concentration	Predicted t_i (min)	Predicted $t_{exposure}$ (min)
CO	23,000 ppm (a)	1.5	3.5
HCN	400 ppm	1.7	7.3
HCl	5000 ppm	74.0	22.0
HF	5988 ppm	29.1	6.2
HBr	1249 ppm	357.0	∞
Acrolein	100 ppm (b)	∞	51.2
CO_2	12% (c)	0.94	No Lethality Data
O_2	3%	0.57 (34 s)	No Lethality Data
NO_2	231 ppm (d)	∞	24.4
SO_2	391 ppm (d)	———	716.0
Temperature	>700°C (1292°F)	0.02 (1 s)	No Lethality Data

(a) Estimate- CO analyzer offscale at 2%
(b) Estimate- based on semiquantitative GC Analysis
(c) Estimate- CO_2 analyzer offscale at 10%
(d) Projected from NBS Smoke Chamber data.

It can be seen that in terms of maximum concentrations, the major incapacitative hazards are CO, HCN, CO_2, O_2, and heat (t_i < 5 minutes). The major LC_{50} hazards are CO, HCN, and heat ($t_{exposure}$ < 5 minutes).

It can also be seen from this table that the narcotic gases (sleep inducing gases) CO, HCN, CO_2, and low O_2 have lower t_i's than $t_{exposure}$. The irritant gases, in contrast, have lower $t_{exposure}$ than t_i's.

PREDICTION OF TIME TO INCAPACITATION FOR FULL-SCALE FIRE TESTS.

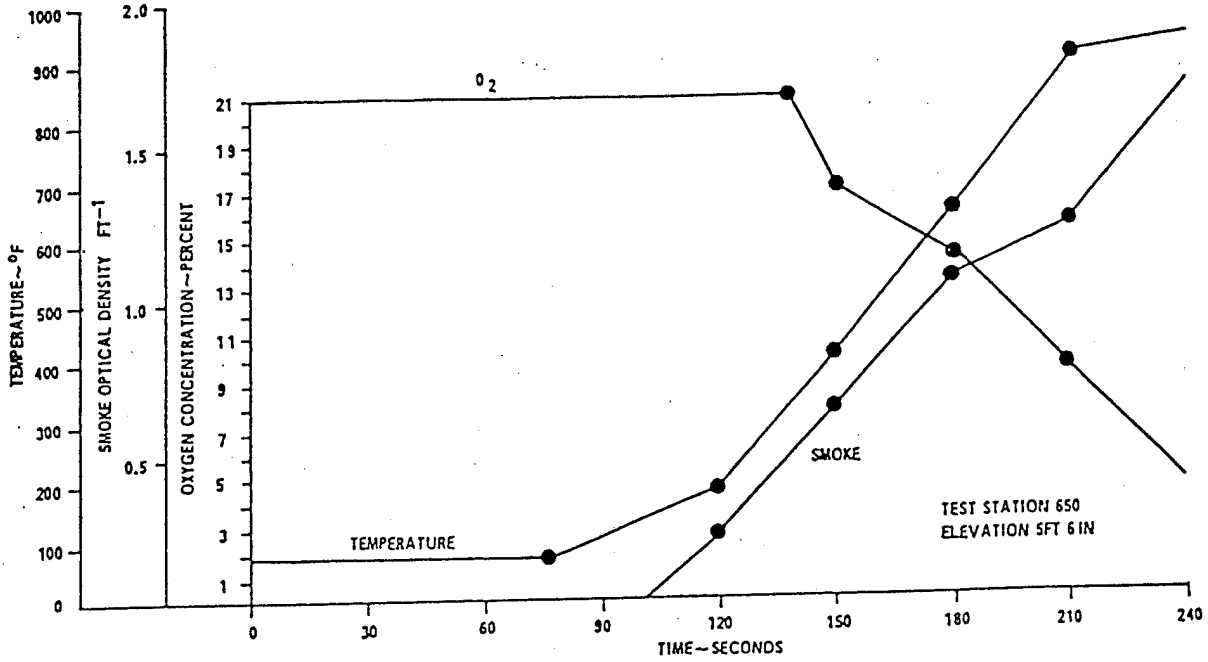
The new FAA model was exercised for two full-scale fire tests conducted at the FAA Technical Center.

C-133 TEST 8135[81,83]. Gas and temperature data for test station 650, elevation 5′6″ in the FAA C-133 Fire Test Article is illustrated in figures 55(a) and 55(b). Pie charts showing the contribution of the various hazards at the point in time when $FED_I = 1$ are illustrated in figure 56. The three pie charts show the new model before the RMV correction, the new model with the RMV correction, and the old model. It is clear that HF and HCl contributes far less to incapacitation with the new model. The old model overstated the incapacitative hazard of these gases. The RMV correction (increased uptake due to CO_2) in the new model results in a greater gas contribution to the FED_I and a slightly shorter t_i.

Wide-Body (C-133) Test 8701, 30 Jul 87 - Final Test[84]. The gas data for test station 880, elevation 3′ 6″ is illustrated in figure 57. No HCN data was taken. The thermal fractional effective dose plotted as a function of time is illustrated in figure 58. The thermal hazard at this height is far less than the thermal hazard at 5′6″ due to the effect of buoyancy manifesting significant thermal gradients. Pie charts for $FED_I = 1$ before and after the RMV correction (V_{CO_2}) are illustrated in figure 59. Toxic gases are the primary contributor to the total FED. The RMV correction shifts t_i from 311 seconds to 276 seconds. Incapacitation is dictated by the CO_2 enhanced inhalation of CO. It is interesting that although the occurrence of flashover generates a rapid increase of hazards inside the cabin, including high temperatures, at some cabin locations (afar from the fire origin, near the floor) survival will be determined by toxic gas exposures.

Wide-Body TC-10 Test[85]. A typical survival curve for a wide-body fuselage based on the FED_I model is illustrated in figure 60. This is based on a full-scale aircraft cabin fire test conducted in a modified DC-10 fuselage.

(a)



(b)

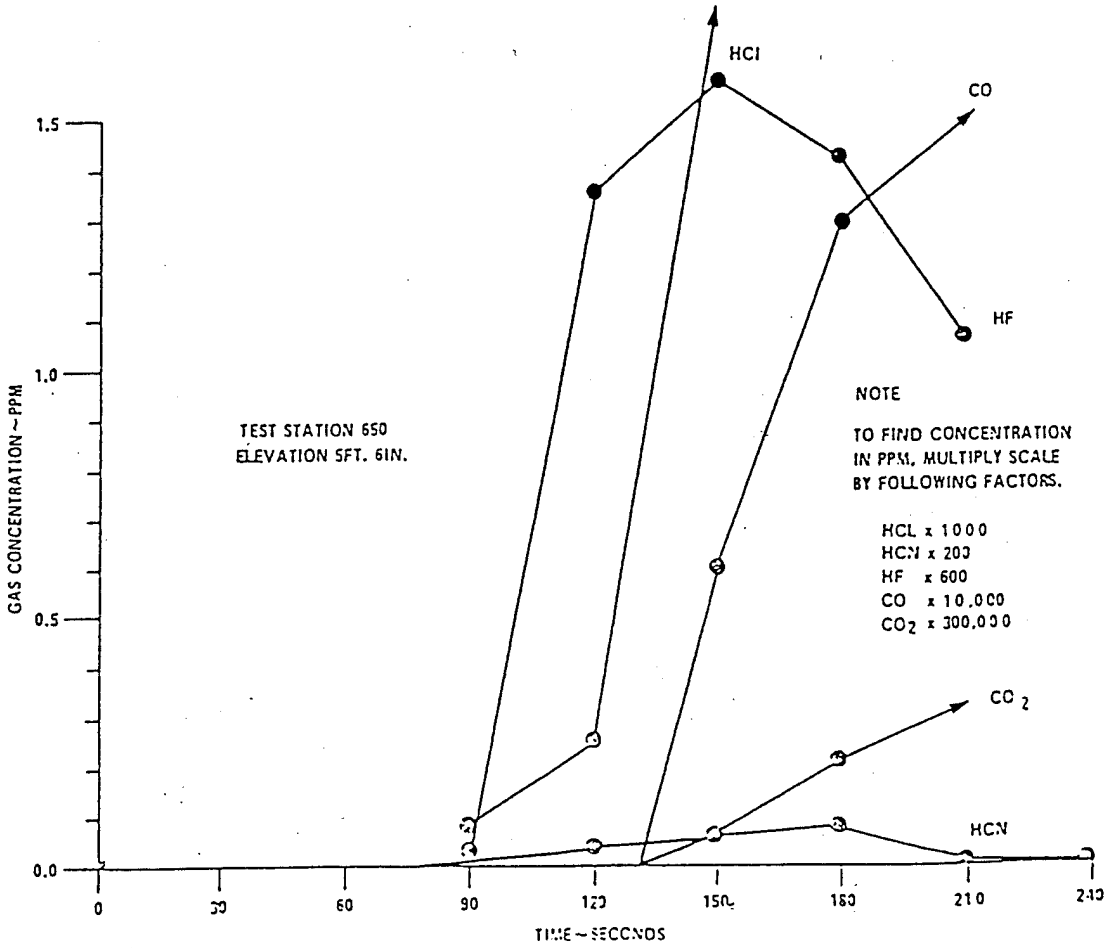


FIGURE 55. HAZARDS IN C-133 AFT CABIN PRODUCED BY BURNING INTERIOR MATERIALS - TEST 8135[81]

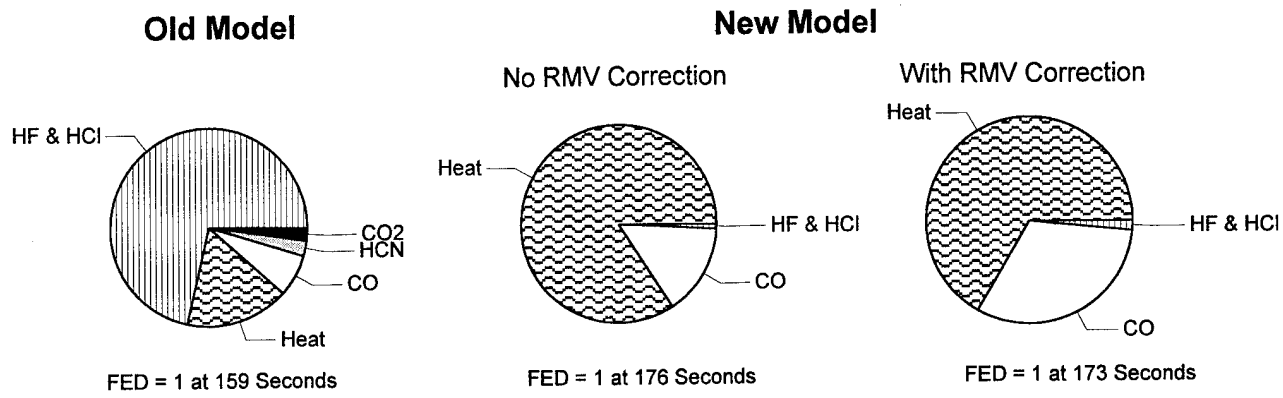


FIGURE 56. FRACTIONAL EFFECTIVE DOSE IN AFT CABIN USING OLD AND NEW SURVIVAL MODELS - TEST 8135

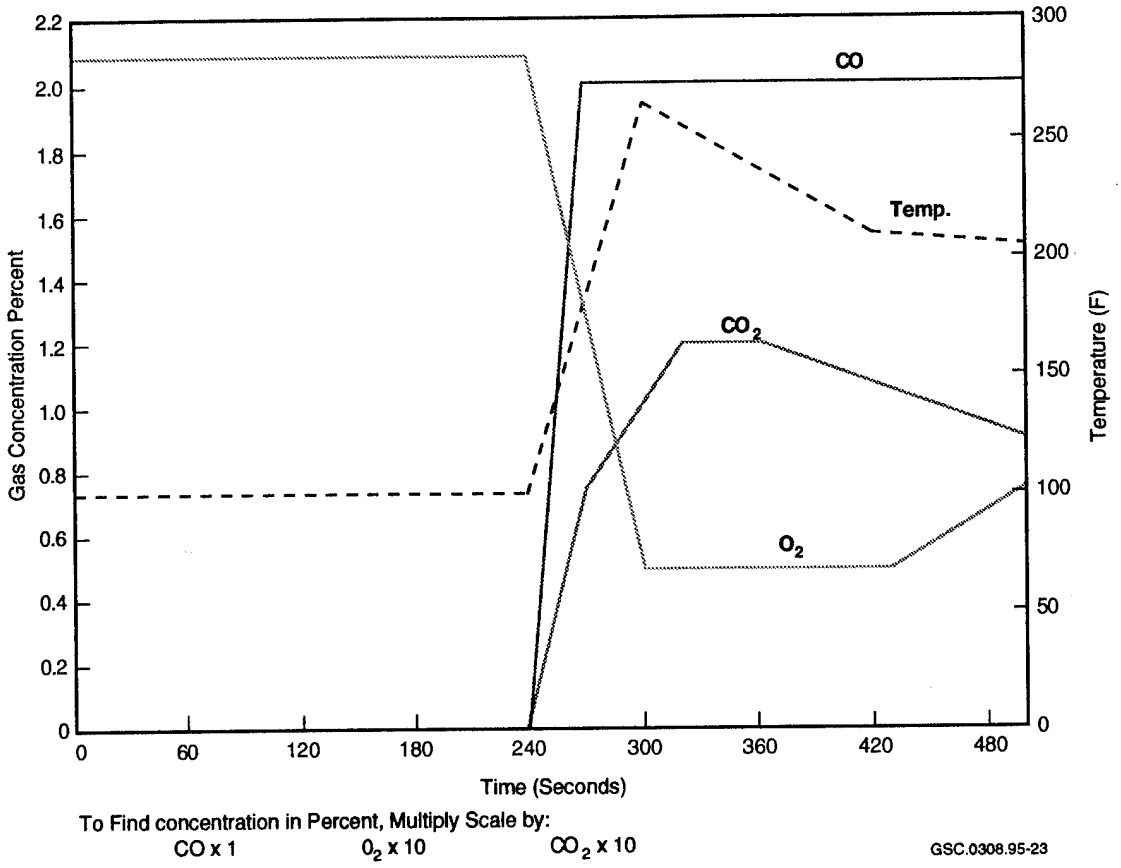


FIGURE 57. HAZARDS IN C-133 AFT CABIN PRODUCED BY BURNING INTERIOR MATERIALS - TEST 8701

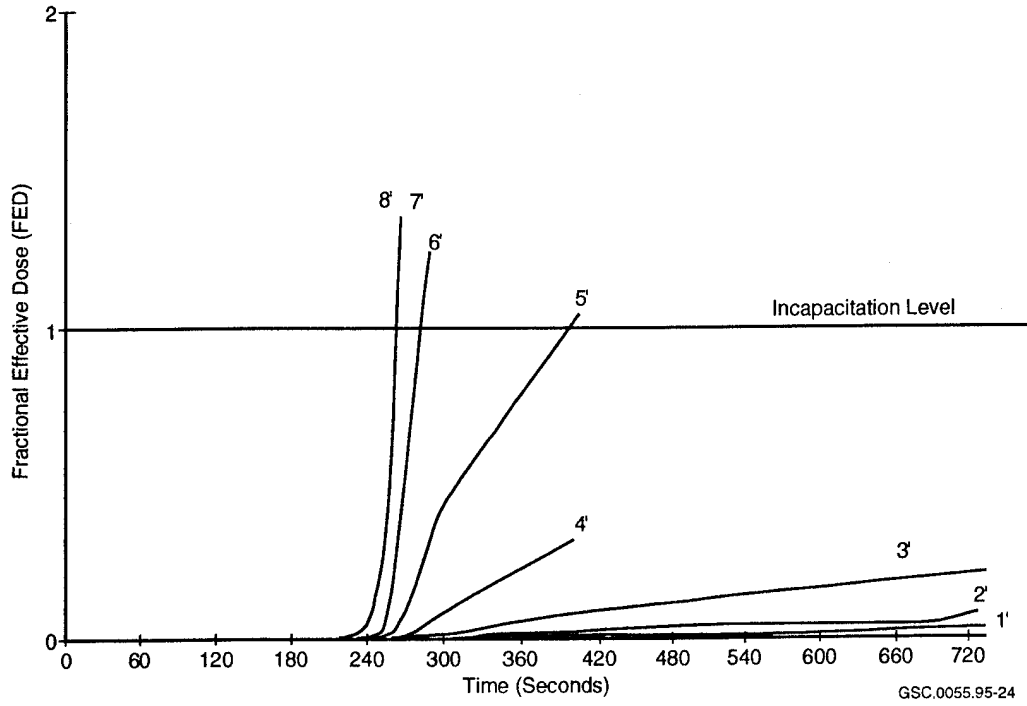


FIGURE 58. THERMAL FRACTIONAL EFFECTIVE DOSE AT VARIOUS CABIN HEIGHTS AT C-133 STATION 880 - TEST 8701[84]

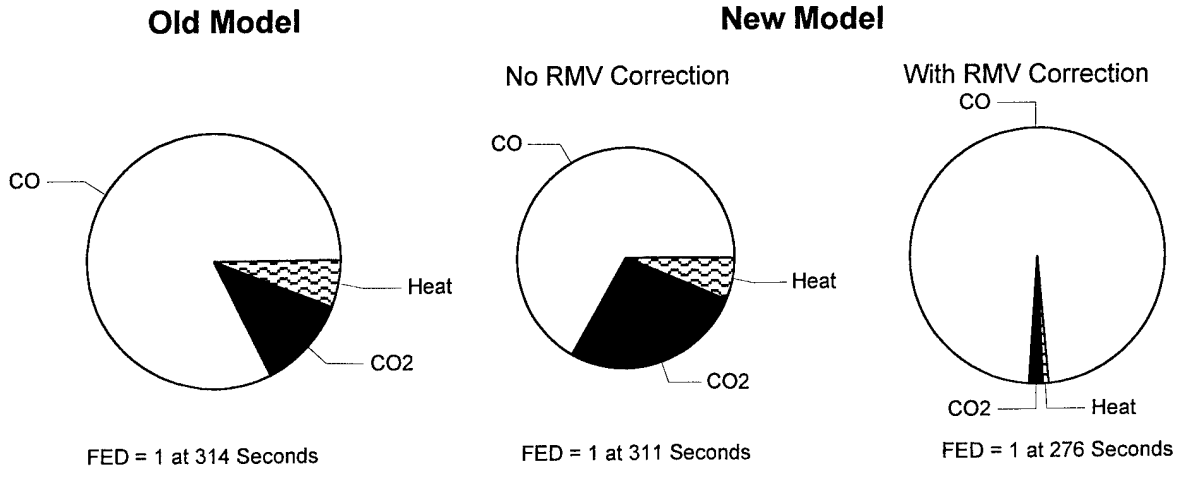


FIGURE 59. FRACTIONAL EFFECTIVE DOSE IN C-133 AFT CABIN USING OLD AND NEW SURVIVAL MODELS - TEST 8701, STATION 880 AT 3 FOOT 6 INCH HEIGHT

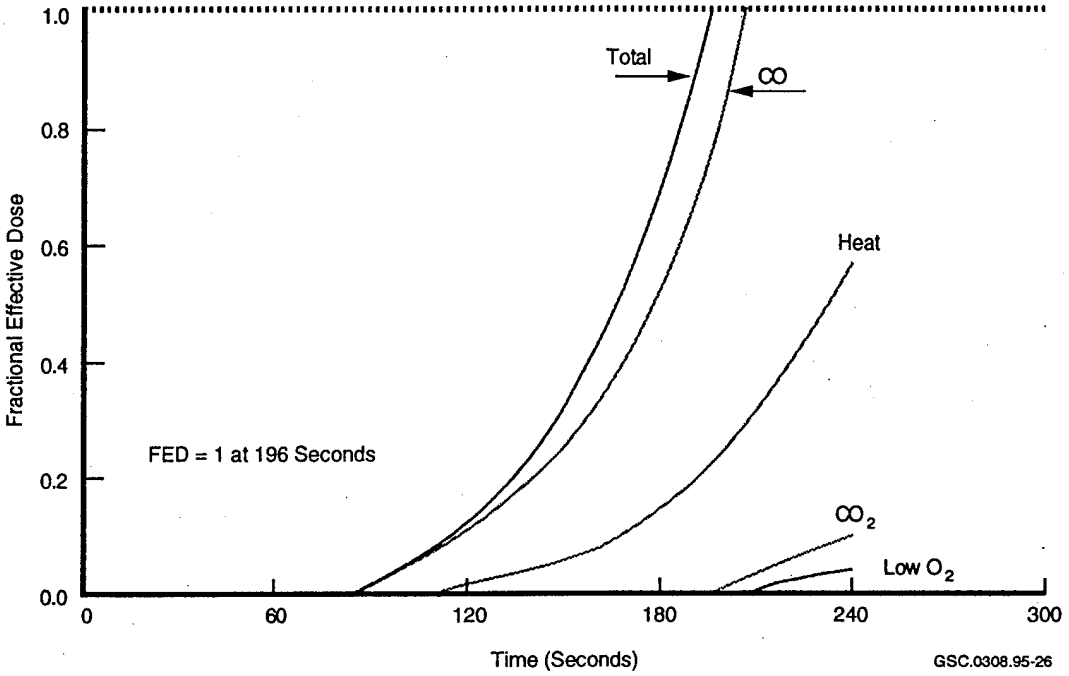


FIGURE 60. TYPICAL SURVIVAL CURVE FOR A WIDE-BODY FUSELAGE BASED ON FED_I MODEL

SUMMARY OF RESULTS

1. Regression equations have been developed based on experimental animal data relating lethal and incapacitative exposure times to a wide range of gas concentrations and temperatures for a variety of species and activity levels.

2. FED_I and FED_L survival models were created by selecting the regression equations judged to best model the human escaping from an aircraft cabin fire.

3. The new FED model considers both the incapacitative and postexposure lethal effect of fire gases. It is based on animal exposure data over a range of concentrations and on human or primate data for CO, CO_2, O_2, HCN, HCl, acrolein, NO_2, SO_2 and heat. It also accounts for the increased ventilation and increased uptake of other gases caused by the presence of CO_2.

4. The incapacitating CO and HCN concentrations for rats are 1.4 times greater for restrained rats than for active rats using Crane's motor-driven rotating wheel protocol. This holds for t_i's ranging from 5 to 60 minutes.

5. Experimental rat data for CO in the presence of 5% CO_2 results in a LC_{50} 1.2 to 1.6 times less than predicted for CO alone. This holds for concentrations 3100 to 11,700 ppm CO. This decrease in LC_{50} is not as great as the predicted RMV increase for rats due to 5% CO_2, a factor of 2.2.

6. Experimental rat data for NO_2 in the presence of 5% CO_2 results in a 30-minute LC_{50} 2.22 times less than predicted for NO_2 alone. This decrease in LC_{50} matches the predicted RMV increase for rats, a factor of 2.2.

7. The RMV increase due to the presence of CO_2 is species dependent.

8. An evacuation study of young men performing a maximum expected evacuation workload showed the mean increase in RMV relative to at rest to be a factor of 2.2 to 2.4.

9. The minute volume increases by the same factor as the oxygen consumption increases (escape task vs. rest) in aircraft evacuation studies of young men performing the maximum expected evacuation workload.

10. The Time of Useful Consciousness of man at rest as a function of oxygen concentrations at sea level has been predicted based on altitude data and agrees with limited normobaric low-oxygen data.

11. Incapacitating CO_2 concentrations are much lower for man than rats.

12. FED_Ls have been shown to be additive for the following gas mixtures for rats: CO + HCN, CO + HCl.

13. Baboons exposed to HCl alone have an increase in RMV. This contrasts to a decrease in the RMV of rodents exposed to HCl alone.

14. Rats exposed to HCl in combination with CO have a longer t_i than when no HCl is present.

CONCLUSIONS

1. Rat test data for NO_2 indicates that the enhanced uptake of NO_2 in the presence of 5% CO_2 results in an LC_{50} 2.2-fold lower. This enhanced uptake matches the predicted RMV increase due to 5% CO_2. One may conclude from this that the other water soluble gases expected to be quantitatively absorbed by the body may also result in LC_{50}s and C_is reduced by the V_{CO_2} factor. Other water soluble gases include HF, HCl, HBr, SO_2, and HCN.

2. Rat test data for CO indicates that the enhanced uptake of CO in the presence of 5% CO_2 results in an LC_{50} 1.2 to 1.6 times less than predicted for CO alone. This falls short of the rat RMV increase of 2.2 due to 5% CO_2. One may conclude from this that the toxicity of CO in presence of CO_2 is over-estimated if V_{CO_2} is used as a multiplicative factor, as is done in the new FED model. Lacking animal exposure data for a full concentration range of CO and CO_2 mixtures, no correction can be made for this effect.

3. Since the RMV increase in the presence of a given concentration of CO_2 is species dependent and since the uptake of other toxic gases also increases in the presence of CO_2, care must be taken in selecting an animal species for toxicity testing of fire gases. For example, the rat and guinea pig do not model human toxic response to a mixture of fire gases with a high CO_2 concentration.

4. While HCl can protect the rat from exposure to hazards such as CO by reducing uptake and delaying the time to incapacitation, HCl enhances uptake in baboons and may shorten the time to incapacitation. Rodents may be an inappropriate species for toxicity testing of gas mixtures containing HCl. Human survival models based on rat toxicity data may not be valid for mixed gases containing HCl.

CAVEATS

Weaknesses of this model include:

1. This model quantifies the thermal threat assuming isothermal exposure. In an actual fire the temperatures at head level are far higher than at waist level.

2. Water is a decomposition product of combustion and the effect of the inhalation of large volumes of hot, moist air is not accounted for in this model.

3. The radiative component of heat was not considered in this model.

4. There is a wide distribution of animal response for both incapacitation and lethality data as well as enhanced uptake in the presence of CO_2. The FED prediction is an average based on a large population of subjects.

5. The low-oxygen FED_I model is based on men at rest. Increased oxygen extraction at higher activity levels may result in significantly higher FED_Is for oxygen.

6. The FED model was based on healthy subjects. Survival of people with impaired cardiovascular and/or pulmonary systems is not addressed in this model.

7. FED's for hazard levels outside the calibration range are not as reliable.

8. The model is as good as the validity of the assumptions: (a) additivity of the hazards and (b) increased uptake of other gases matches the increase in RMV due to the presence of CO_2.

REFERENCES

1. Purser, D., "Toxicity Assessment of Combustion Products," SFPE Handbook of Fire Protection Engineering, First Edition, Chapter 14, 200–245, 1988, Copyright 1988, Society of Fire Protection Engineers, Boston, Massachusetts.

2. Stewart, R., Peterson, J., Baretta, E., Dodd, H., and Herrmann, A., Archives of Environmental Health, Volume 21, p. 154 (1970).

3. Stewart, R., Peterson, J., Fisher, T., Hosko, M., Baretta, E., Dodd, H., and Herrmann, A., Archives of Environmental Health, Volume 26, p. 1 (1973).

4. Documentation of the Threshold Limit Values and Biological Exposure Indices, American Conference of Governmental Industrial Hygienists, Sixth Edition, Copyright 1991, Cincinatti, Ohio.

5. Kaplan, H., Grand, A., Switzer, W., Mitchell, D., Rogers, W., Hartzell, G., "Effects of Combustion Gases on Escape Performance of the Baboon and the Rat," Journal of Fire Sciences, Volume 3, 228–244, July/August 1985, Copyright 1985, Technomic Publishing Co., Lancaster, Pennsylvania.

6. Hartzell, G., Priest, D., and Switzer, W., "Modeling of Toxicological Effects of Fire Gases: II. Mathematical Modeling of Intoxication of Rats by Carbon Monoxide and Hydrogen Cyanide," Journal of Fire Sciences, Volume 3, Number 2, pp. 115-128, March/April 1985, Copyright 1985, Technomic Publishing Co., Lancaster, Pennsylvania.

7. Crane, C., Sanders, D., Endecott, B., "INHALATION TOXICOLOGY: IX. Times to Incapacitation for Rats Exposed to Carbon Monoxide Alone, to Hydrogen Cyanide Alone, and to Mixtures of Carbon Monoxide and Hydrogen Cyanide," Federal Aviation Administration, Civil Aeromedical Institute, Report Number DOT/FAA/AM-89/4 January 1989.

8. Purser, D., Berrill, K., "Effects of Carbon Monoxide on Behavior in Monkeys in Relation to Human Fire Hazard," Archives of Environmental Medicine, Volume 38, Number 5, 308–315, September/October 1983.

9. Sakurai, T., "Toxic Gas Tests of Several Pure Gases and Mixed Gases using Mice" Journal of Fire Sciences, Volume 7, January/February 1989, Copyright 1989 by Technomic Publishing Co., Lancaster Pennsylvania.

10. Hartzell, A., Grand, A., and Switzer, W., "Modeling of Toxicological Effects of Fire Gases: VI. Further Studies on the Toxicity of Smoke Containing Hydrogen Chloride," Journal of Fire Sciences, 5, 368–391, November/December 1987, Copyright 1987, Technomic Publishing Co., Lancaster, Pennsylvania.

11. Levin, B., Gruman, J. Paabo, M., Baier, L., Holt, T., "Toxicological Effects of Different Time Exposures to the Fire Gases: Carbon Monoxide or Hydrogen Cyanide or to Carbon Monoxide Combined with Hydrogen Cyanide or Carbon Dioxide," 9th Joint Panel Meeting of the U.S.-Japan Government Cooperative Program on Natural Resources (UJNR) Panel on Fire Research and Safety, National Bureau

of Standards, NBSIR 88-3753, pp. 368-383, April 1988. And Society of the Plastics Industry (SPI) Polyurethanes 88, 31st Annual Technical/Marketing Conference, 1988, Philadelphia, PA, pp. 240-248.

12. Purser, D., "A Bioassay Model for testing the Incapacitating Effort of Exposures to Combustion Product Atmospheres using Cynomolgus Monkeys," Journal of Fire Sciences, Volume 2, January/February 1984, 20–37.

13. Purser, D., Grimshaw, P., and Berrill, K., "Intoxication by Cyanide in Fires: A Study in Monkeys using Polyacrylonitrile," Archives of Environmental Health, Volume 39, Number 6, 394–400, November/December 1984.

14. Hartzell, G., Advances in Combustion Toxicology, Volumes 1 and 2, 1988, Technomic Publishing Co., Lancaster, Pennsylvania.

15. Haber, F., Funf Vortrange aus den jaren 1920-1923, Verlag von Julius Springer, Germany 1924.

16. Kimmerle, G., Journal of Combustion Toxicology, Volumes 1 and 4, 1974.

17. Kaplan, H., Grand, A., Rogers, W., Switzer, W., Hartzel, G., "A Research Study of the Assessment of Escape Impairment by Irritant Combustion Gases in Postcrash Aircraft Fires," Federal Aviation Administration Report Number DOT/FAA/CT-84/16, 1984.

18. Kaplan, H., Anzueto, A., Switzer, W., Hinderer, R., "Effects of Hydrogen Chloride on Respiratory Responses and Pulmonary Function of the Baboon," Journal of Toxicology and Environmental Health, Volume 23, 473–493, 1988, Copyright 1988 by Hemisphere Publishing Co., New York, New York.

19. Hartzell, G., Packham, S., Grand, A., Switzer, W., "Modeling of Toxicological Effects of Fire Gases: III. Quantification of Postexposure Lethality of Rats from Exposures to HCl Atmospheres," Journal of Fire Sciences, Volume 3, No. 3, pp. 195-207, May/June 1985, Copyright 1985, Technomic Publishing Co., Lancaster, Pennsylvania.

20. Crane, C., Sanders, D., Endecott, B., Abbott, J., "Inhalation Toxicology: IV. Times to Incapacitation and Death for Rats Exposed Continuously to Atmospheric Hydrogen Chloride Gas," Federal Aviation Administration, Civil Aeromedical Institute, Report Number FAA-AM-85-4, May 1985.

21. Higgins, E., Fiorica, V., Thomas, A., Davis, H., "The Acute Toxicity of Brief Exposures to HF, HCl, NO_2 and HCN Singly and in Combination with CO. Federal Aviation Administration, Civil Aeromedical Institute, Report Number FAA-AM-71-41, 1971.

22. Wohlslagel, J., Dipasquale, L., Vernot, E., "Toxicity of Solid Rocket Motor Exhaust: Effects of HCl, HF, and Alumina on Rodents," Journal of Combustion Toxicology, Volume 3, February 1976, 61–70.

23. Skogg, E., "A Toxicological Investigation of Lower Aliphatic Aldehydes: 1. Toxicity of Formaldehyde, Acetaldehyde, Propionaldehyde, and Butyraldehyde; as well as of Acrolein and Crotonaldehyde" Axta Pharmacology, 1950, Volume 6, 299–318.

24. Pattle, R.E., Cullumbine, H., "Toxicity of Some Atmospheric Pollutants," British Medical Journal, 1956, Volume 2, 913–916.

25. Bouley G., Dabreuil A., Godin J., Boisset M., Boudence C.L., "Phenomena of Adaptation in Rats Continuously Exposed to Low Concentrations Acrolein," Ann. Occup. Hyg. 1976, Volume 19, 27–32.

26. Murphy S.D., Davis H.V., Ulrich C.E., "Functional Effects of Acrolein Exposure," Fed. Proc. 1962, Volume 21, p. 221.

27. Crane, C., Sanders, D., Endecott, B., Abbott, J., "INHALATION TOXICOLOGY: VII. Times to Incapacitation and Death for Rats Exposed Continuously to Atmospheric Acrolein Vapor." Report Number DOT/FAA/AM-86/5. Washington, D.C.: Federal Aviation Administration, Office of Aviation Medicine, 1986.

28. Creasia, D., "Pathogenesis of Nitrogen Dioxide-Induced Respiratory Lesions in Reference to Respiratory Clearance of Inhaled Particulates," Journal of Toxicology and Environmental Health, 8: 857–871, 1981.

29. Carson, T., Rosenholtz, M., Wilinski, F., and Weeks, M., "The Responses of Animals Inhaling Nitrogen Dioxide for Single Short Term Exposures," Am. Ind. Hyg. Assoc. J., 23: 457–462, 1962.

30. Gray, E., Patton, F., Goldberg, S., Kaplan, E., "Toxicity of the Oxides of Nitrogen: II. Acute Inhalation Toxicity of Nitrogen Dioxide, Red Fuming Nitric Acid, and White Flaming Nitric Acid," A.M.A. Archives of Industrial Hygiene and Occupational Medicine, now known as Archives of Environmental Health 10: 418–422, 1954, Copyright 1954 by Heldref Publication, Washington, D.C.

31. Higgins E., Fiorica V., Thomas A., Davis, H., "The Acute Toxicity of Brief Exposures to HF, HCl, NO_2 and HCN Singly and in Combination with CO. Federal Aviation Administration, Civil Aeromedical Institute, Report Number FAA-AM-71-41, 1971.

32. Levin, B., Paabo, Highbarger, L., Eller, N., "Synergistic Effects of Nitrogen Dioxide and Carbon Dioxide Following Acute Inhalation Exposures in Rats," NISTIR 89-4105, National Institute of Standards and Technology, Gathiersburg, Maryland 1989.

33. Bitron, M., Aharonson, E., "Delayed Mortality of Mice Following Inhalation of Acute Doses of CH_2O, SO_2, Cl_2, and Br_2," American Industrial Hygiene Journal (39) February 1978.

34. King, B., "High Concentration Short Time Exposures and Toxicity" Journal Industrial Hyg. Toxicol., 31, 365–375, 1949.

35. Haldane, Respiration, Yale University, New Haven, Connecticut 1922.

36. Schute, J., Archives Environmental Health, 8, 427 1964.

37. Blockley, W., Bioastronautics Data Book, NASA SP-3006, pp. 5-17, pp. 103-132, 1964.

38. Nevison, T. "Letter Report to the Garrett Corporation," The Lovelace Foundation, Albuquerque, New Mexico, January 25, 1962.

39. Schaeffer, K., "A Concept of Triple Tolerance Limits Based on Chronic Carbon Dioxide Toxicity Studies," Aerospace Medicine, 32, 197–204, 1961.

40. Lambertson, C., Anesthesiology, 31, 642, 1962.

41. Comroe, J., Forster, R., Dubois, A., Briscoe, W., and Carlsen, E., "The Lung," 52–53, Year Book Medical Publishers, Inc., Chicago, Illinois, 1962.

42. Altman, P., and Ditter, D., ed., "Respiration After Acute Exposure to High Carbon Dioxide Concentrations: Mammals," Environmental Biology, Federation of American Societies for Experimental Biology, Bethesda, Maryland, 1966.

43. Brown, E., "The Physiological Effects of High Concentrations of Carbon Dioxide," U.S. Naval Medical Bulletin, Volume 28, 721–734, October 1930.

44. Davies, H., Haldane, J., and Kennaway, E., "Experiments on the Regulation of the Blood's Alkalinity," J. of Physiology, Volume 54, 32–45, 1920.

45. Brown, E., "The Value of High Oxygen in Preventing the Physiological Effects of Noxious Concentrations of Carbon Dioxide," U.S. Naval Medical Bulletin, Volume XXVIII, July 1930, Number 3.

46. White, Clayton S., "Estimated Tolerance of Human Subjects to Various CO_2-Time Concentrations," Lovelace Clinic Project 200, Report Number 2, July 27, 1948. Lovelace Medical Library, Albuquerque, New Mexico.

47. Hill, L. and Flack, M., "The Effects of Excess of CO_2 and Want of O_2 on the Respiration and Circulation," Journal Physiology, 37, pp. 77-111, 1908.

48. Haldane, J., and Smith, J., "The Physiological Effects of Air Vitiated by Respiration." Journal of Pathology and Bacteriology, Vol. 1, pp. 168-186, 1892.

49. Dripps, R.D. and Comroe, J.H., "The Respiratory and Circulatory Response of Normal Man to Inhalation of 7.6 and 10.4 Percent CO_2 with a Comparison of the Maximal Ventilation Produced by Severe Muscular Exercise, Inhalation of CO_2 and Maximal Voluntary Hyperventilation," American Journal of Physiology, Vol. 149, pp. 43-51, 1947.

50. Case, E.M. and Haldane, J.B., "Human Physiology under High Pressure: Effects of Nitrogen, Carbon Dioxide, and Cold," Journal Hyg., Vol. 41, pp. 225-249, 1941.

51. Consolazio, W.V., Fisher, M.B., Pace, J., Pecora, L.J., Pitts, G.C., and Behnke, A.R., "Effects on Man of High Concentrations of Carbon Dioxide in Relation to Various Oxygen Pressures During Exposures as Long as 72 Hours." American Journal of Physiology, Vol. 151, pp. 479-503, 1947.

52. Levin, B., Paabo, M., Gurman, J., Harris, S., Braun, E., "Toxicological Interactions Between Carbon Monoxide and Carbon Dioxide," Toxicology, 47, 135–164, 1987, Copyright 1987, Elsevier Scientific Publishers Ireland Ltd., Limerick, Ireland.

53. Herpol, C., Minno, R., Van Outryve, E., "Biological Evaluation of the Toxicity of Gases Produced under Fire Conditions by Synthetic Materials. Part I: Methods and Preliminary Experiments Concerning the Reaction of Animals to Simple Mixtures of Air and Carbon Dioxide or Carbon Monoxide," J. Combustion Sci. Technol., 12, 217–228, 1976.

54. Luft, U., in Handbook of Physiology, Section 3: Respiration, ed. by Fenn, W. and Rahn, H., 1099–1145, American Physiology Society, Washington, 1965.

55. Shukitt, B., Burse, R., Bandaret, L., Knight, D., Cymerman, A., "Cognitive Performance, Mood States, and Altitude Symptomatology in 13 Oxygen Environments," U.S. Army Research Institute of Environmental Medicine, Natick, Massachusetts, Report Number T18-88, June 1988.

56. Knight, D., Luria, S., Socks, J., Rogers, W., "Effect of Nitrogen-Based Fire Retardant Atmospheres on Visual and Mental Performance," 9th International Symposium on Underwater and Hyperbaric Physiology, Undersea and Hyperbaric Medical Society, Bethesda, Maryland, 1987.

57. Luria, S., and Knight, D., "Scotopic Sensitivity with 10% Oxygen," Naval Submarine Medical Research Laboratory, Submarine Base, Groton, Connecticut, Report Number 1097, July 1987.

58. Slobodnik, B., Wallick, M., Chimak, J.M., "Effectiveness of Oxygen-Nitrogen Gas Mixtures in Inducing Hypoxia at 1 ATA," Navy Experimental Diving Unit Report No. 04-91, Department of the Navy, Panama City, Florida, June 1991.

59. Ersting, J., King, P., eds., Aviation Medicine, Second Edition, Boston, Butterworths, Copyright 1988, p. 56.

60. U.S. Standard Atmosphere, 1962, Washington, D.C., U.S. Government Printing Office, 1963, p. 9.

61. Gall, C.F., in: Van Liere, E., Stickney, J. (ed.), Hypoxia, Chicago: The University of Chicago Press, 1963, 308–311.

62. Izraeli, S., Avgar, D., Glickson, M., Shochat, I., Glovinsky, Y., Ribak, J., "Determination of the 'Time of Useful Consciousness' (TUC) in Repeated Exposures to Simulated Altitude of 25,000 Feet," Aviation, Space and Environmental Medicine, 1103–1105, November 1988.

63. Levin, B., Paabo, M., Gurman, J., Harris, S., "Effects of Exposures to Single or Multiple Combinations of the Predominant Toxic Gases and Low Oxygen Atmospheres Produced in Fires," Fundamental and Applied Toxicology, 9, 236 1987.

64. Matijak-Schaper, M., Alarie, Y., "Toxicity of Carbon Monoxide, Hdrogen Cyanide and Low Oxygen," Journal of Combustion Toxicology, Volume 9, 21–61, February 1982.

65. Crane, C., "Human Tolerance Limit to Elevated Temperature: An Empirical Approach to the Dynamics of Acute Thermal Collapse" Federal Aviation Administration, Memorandum Report No. ACC-114-78-2, May 1978.

66. Blockley, W.V., in Biology Data Book, Federation of American Societies for Experimental Biology, Bethesda, Maryland, 781–784, 1973.

67. Hofer, R., "On The Effect of Gas Mixtures" (in German), Jaunyn-Schmiedebergs Archiv fur Experimentelle Pathologie und Pharmakologie 111: 183–205, 1926.

68. Tsuchiya, Y., "On the Unproved Synergism of the Inhalation Toxicity of Fire Gas," Journal of Fire Sciences, 4: 346–354, 1986.

69. Hartzell, G.E., Switzer, W.G., and Priest, D.N., "Modeling of Toxicological Effects of Fire Gases: V. Mathematical Modeling of Intoxication of Rats by Combined Carbon Monoxide and Hydrogen Cyanide Atmospheres," Journal of Fire Sciences, 3, Number 5, 330–342, September/October 1985.

70. Switzer, Walter G., "Investigation of the Interactive Effects between Carbon Monoxide and Hydrogen Cyanide on Time-to-Incapacitation for Fire Hazard Modeling," Final Report, Southwest Research Institute Number 01-9316, April 1986.

71. Wong, K., Alarie, Y., "A Method for Repeated Evaluation of Pulmonary Performance in Unanesthetized, Unrestrained Guinea Pigs and its Application to Detect Effects of Sulfuric Acid Mist Inhalation," Toxicology and Applied Pharmacology, Volume 63, 72–90, 1982, Copyright 1982 by Academic Press, Orlando, Florida.

72. Chapin, J., "Ventilatory Response of the Unrestrained and Unanesthetized Hamster to CO_2. American Journal of Physiology, Vol. 179, pp. 146-148, 1954.

73. Lai, Y., Lamm, J., Hildebrandt, J., "Ventilation During Prolonged Hypercapnia in the Rat," Journal of Applied Physiology: Respiration. Environment Exercise Physiology, Volume 51, 78 1981, Copyright 1981, American Physiological Society, Bethesda, Maryland.

74. Levin, B.C. "Determination of the Toxicological Effects of Fire Gases, Alone and in Various Combinations, for Use in Toxic Hazard Assessment Computer Models," unpublished paper presented at the third expert meeting of the Canada-Japan-U.S.A. cooperative study on fire gas toxicity, Ottawa, Canada, October 1984. See also Chemical and Engineering News, 63 (20): 3, May 20, 1985.

75. Crane, C. "Are the Combined Toxicities of CO and CO_2 Synergistic?," Journal of Fire Sciences, 3: 143–144, 1985.

76. Gibbs, F., Gibbs, E., Lennox, W., Nims, L., Journal of Aviation Medicine, 14, 250, 1943.

77. Karl, A., McMillan, G., Ward, S., Kissen, A., Souder, M., "Effects of Increased Ambient CO_2 on Brain Tissue Oxygenation and Performance in the Hypoxic Rhesus,"Aviation, Space, and Environmental Medicine, Volume 49, 984–989, August 1978.

78. Root, W., in Handbook of Physiology Section 3: Respiration, ed. by W. Fenn and H. Rahn, 1087–1098, American Physiological Society, Washington, D.C., 1985.

79. Espisito, F., Alarie, Y., "Inhalation Toxicity of Carbon Monoxide and Hydrogen Cyanide Gases Released During the Thermal Decomposition of Polymers," Journal of Fire Sciences, Volume 6, May/June 1988, 195–242.

80. Ross, J., Watt, S., Henderson, G., "Ventilation and Oxygen Uptake During Escape from a Civil Aircraft," Ergonomics, Volume 33, Number 1, 13–24, 1990, Copyright 1990 by Taylor and Francis, Ltd., London, New York, Philadelphia.

81. Sarkos, C., Hill, R., and Howell, W., "The Development and Application of a Full-Scale Wide-Body Test Article to Study the Behavior or Interior Materials During Post-Crash Fuel Fire,"AGARD Lecture Series Number 123 on Aircraft Fire Safety, AGARD-LS-123, June 1982.

82. Spieth, H., Gaume, J., Luoto, R., Klinck, D., "A Combined Hazard Index Fire Test Methodology for Aircraft Cabin Materials," Federal Aviation Administration, FAA Technical Center Report Number DOT/FAA/CT-82/36-1, April 1982, available from National Technical Information Service, Springfield, VA 22161.

83. Hill, R.G., Brown, L.J., Speitel, L.C., Johnson, G.R., Sarkos, C., "Aircraft Seat Fire Blocking Layers—Effectiveness and Benefits Under Various Scenarios, Federal Aviation Administration, FAA Technical Center, Report No. DOT/FAA/CT-83/43, February 1984. Available from National Technical Information Service, Springfield, VA 22161.

84. Sarkos C., Hill R., "Characteristics of Transport Aircraft Fires Measured by Full-Scale Tests," AGARD Lecture Series Number 467 in Aircraft Fire Safety, 1990.

85. Marker, T., "Wide-Body Cabin Water Spray Optimization Tests," Federal Aviation Administration, FAA Technical Center, Technical Note No. DOT/FAA/CT-TN 93/29, August 1993.

86. Levin, B.C., "The Development of a New Small Scale Smoke Toxicity Test Method and Its Comparison with Real Scale Fire Tests," Toxicology Letters, Vol. 64/65, pp. 257-264, 1992.

87. Levin, B.C., Braun, E., Navarro, M., Paabo, M., "Further Development of the N-Gas Mathematical Model: An Approach for Predicting the Toxic Potency of Complex Combustion Mixtures," In Fire and Polymers II: Material and Tests for Hazard Prevention, Nelson, G.L. Ed.; ACS Symposium Series No. 599; American Chemical Society, Washington, D.C., 1995, Chapter 20, pp. 293-311.

88. Bukowski, R., Jones, W., Levin, B., Forney, C., Stiefel, S., Babrauskas, V., Braun, E., Fowell, A., "Hazard I. Volume I: Fire Hazard Assessment Method," National Bureau of Standards, Center for Fire Research, Gaithersburg, MD, Report No. NBSIR 87-3602, July 1987.

APPENDIX A
DEFINITIONS

Acidosis: A condition in which the pH of the blood is lowered (i.e., becomes more acidic). Respiratory acidosis in fire exposures results from excess carbon dioxide uptake. Metabolic acidosis results from impaired tissue respiration (due to tissue hypoxia) caused by burns or narcosis.

Addition: Two or more toxic substances are considered to exert an additive effect when they act in concert such that the effect in combination is greater than the effect of either substance acting alone, but not greater than the sum of the effects of either substance acting alone (when they may be said to be directly additive). (See also synergism).

Air: 78% nitrogen, 20.9% oxygen, 0.9% argon, and additional gases in trace concentrations.

Air Hunger: A symptom of CO_2 exposure in humans.

Alveoli: Air sacs at the termination of the bronchi in the lung. It is here that oxygen is absorbed into the blood and carbon dioxide is released into the lung.

Apnea: Temporary suspension of respiration.

Asphyxia: Suffocation, decrease in the oxygen content, and increase in the carbon dioxide content of the blood. This may occur due to laryngeal spasm caused by burns or irritant gasses, or to impairment of breathing or gas exchange in the lung. The term has been extended to include all causes of tissue hypoxia, including exposure to asphyxiant gases (low oxygen concentration due to the excess of any other gas,or exposure to the narcotic gases carbon monoxide and hydrogen cyanide, which produces asphyxia chemically).

Behavioral Effects/Incapacitation: The extent to which exposure to fire products affects the ability or willingness of a subject or experimental animal to perform coordinated movements or tasks, particularly movements or tasks similar to those required to escape from a fire. (See incapacitation.)

Bronchoconstriction: Constriction of the conducting airways in the lung due to the contraction of smooth muscle in the airway walls in response to an agonist or to stimulation of irritant receptors acting through the vagus nerve.

Carboxyhemoglobin (COHb): Combination of carbon monoxide with hemoglobin in the blood, which limits the combination of hemoglobin with oxygen (oxyhemoglobin) and therefore the transport of oxygen in the blood.

Cerebral Depression: Condition in which the electrical activity of the cerebral cortex as revealed in the electroencephalogram consists mainly of slow wave (or delta wave) activity. This is typical of a semiconscious or unconscious state.

Concentration: The amount of a gas in the atmosphere per unit volume of the atmosphere, usually quoted as mass/volume (mg/L or mg/m^3) or volume/volume (ppm or percent)

Dose: The amount of toxicant to which a fire victim or test animal is exposed. The simplest estimation of dose for inhalation toxicology is to multiply the atmosphere concentration (Ct product). A lethal dose may be expressed in terms of the $L(Ct)_{50}$. However, other factors may affect the amount of toxicant actually entering the body.

Dyspnea: Subjective difficulty or distress in breathing.

Edema: Accumulation of an excessive amount of fluid in cells, tissues, or body cavities. Pulmonary edema occurs when a fluid exudate leaks out of blood vessels as a result of inflammation or circulatory insufficiency, and the lung tissue becomes swollen and waterlogged. Further development results in a fluid exuded within the alveolar spaces. This fluid accumulation seriously affects gas exchange in the lung and may be fatal.

Fractional Effective Dose (FED): This is useful in estimating the toxic hazard of a substance at a particular point in time. For a constant concentration of the toxicant, the FED is the dose received up to time t into the exposure divided by the effective Ct dose to cause an effect (e.g., incapacitation or death). The effect is predicted to occur when the FED is equal to one. FEDs can be calculated for exposures of mixed gases with concentrations changing over time. See text.

Haber's Rule: Principle that toxicity in inhalation toxicology depends on the dose available and that the product of concentration and exposure time is constant.

Hazard: A toxic fire hazard exists when a toxic product is present at a sufficient concentration and over a sufficient period of time to cause a toxic effect. A physical fire hazard exists when a physical fire parameter (heat or smoke) is present at an intensity and over a period sufficient to cause injury or seriously inhibit the ability to escape from a fire.

Hemoglobin: The protein in red blood cells that transports 99% of the oxygen carried by the blood stream.

Hyperbaric: Pertaining to pressure of ambient gases greater than 1 atmosphere.

Hypercapnia: Increased blood carbon dioxide concentration.

Hyperthermia (Heat Stroke): An increase in body temperature above 37°C. Hyperthermia is life threatening if the body core temperature or temperature of the blood entering the heart exceeds 42.5°C.

Hyperventilation: Increased rate and depth of breathing (increased respiratory minute volume [RMV]) in response to increased carbon dioxide, hypoxic hypoxia, hydrogen cyanide, exercise, heat, or stimulation of pulmonary irritant receptors.

Hypobaric: Pertaining to pressure of ambient gases less than 1 atmosphere.

Hypoxia: A reduction in the amount of oxygen available for tissue respiration. This can occur in four ways.

Anemic Hypoxia: A condition in which the arterial P_{O_2} is normal, but the amount of hemoglobin available to carry oxygen is reduced and/or the ability to release oxygen to the tissues is impaired. For fire exposures this results mainly from the formation of carboxyhemoglobin following exposure to CO, but an anemic subject would be at increased risk.

Histoxic Hypoxia: A condition in which the amount of oxygen delivered to the tissues is adequate, but due to the action of a toxic agent such as HCN, the tissue cells cannot make use of the oxygen supplied to them.

Hypoxic Hypoxia (low oxygen Hypoxia): A condition in which the P_{O_2} of the arterial blood is reduced as a result of low atmospheric oxygen concentration or impairment of gas exchange in the lung due to bronchoconstriction or respiratory tract damage or disease.

Ischemic Hypoxia: A condition in which the blood flow to a tissue is so low that adequate oxygen is not delivered to it despite a normal P_{O_2} and hemoglobin concentration. This occurs during shock following burns and in cerebral tissue due to alkalosis, or briefly during postural hypotension.

Incapacitation: An inability to perform a task (related to escape from a fire) caused by exposure to a toxic substance or a physical agent in a fire. A distinction is sometimes made between severe physiological incapacitation, in which the subject is unable to move normally, such as might occur in an unconscious or badly burned victim and behavioral incapacitation, such as that caused by visual obscuration or eye irritation from smoke, in which the victim is more or less intact, but still unable to escape from the fire.

Inflammation: A complex series of reactions occurring in blood vessels and adjacent tissues around the site of an injury. The initial reaction is congestion (engorgement of local blood vessels), exudation of fluid into the tissues (edema), and pain. This is followed by a phase of destruction and removal of injured tissue by inflammatory cells and then a phase of repair.

Intoxication: A state in which a subject is adversely affected by a toxic substance. Specifically, the time at which a subject has taken up sufficient narcotic (asphyxiant) gas that he or she behaves like someone severely affected by alcohol.

Irritation and Irritancy: Irritation is the action of an irritant substance; irritancy is the response. This response takes two forms:

Pulmonary (Lung) Irritant: Response occurs when an irritant penetrates into the lower respiratory tract. This may result in breathing discomfort (dyspnea), bronchoconstriction, and an increase in respiratory rate during the fire exposure. In severe cases it is followed after a period (usually of a few hours) by pulmonary inflammation and edema, which may be fatal.

Sensory Irritant: Response occurs when an irritant substance comes in contact with the eyes and upper respiratory tract (and sometimes the skin), causing a painful sensation accompanied by inflammation with lacrimation or mucus secretion. At low concentrations, this adds to the visual obscuration caused by smoke, but at high concentrations the severe effects may have behavioral, and to some extent, physiological incapacitation.

Lacrimation: The production of tears in response to sensory irritation of the eyes.

LC_{50}: Lethal concentration—50 percent. The concentration statistically calculated to cause the deaths of one half of the animals exposed to a toxicant for a specified time. Deaths include post-exposure deaths. It may be expressed as volume/volume (ppm, percent), or mass/volume (mg/L).

$L(Ct)_{50}$: The product of exposure concentration and duration of exposure, causing the death of 50 percent of animals.

Mountain Sickness: Sickness caused by insufficient oxygen at high altitudes marked by shortness of breath and nausea.

Narcosis: Literally "sleep induction" but used in combustion toxicology to describe central nervous system depression causing reduced awareness, intoxication, and reduced escape capability leading to loss of consciousness and death in extreme cases. The narcotic gases CO, HCN, and CO_2 cause narcosis, as does lack of oxygen due to the inhalation of an atmosphere low in oxygen, an impairment in breathing, or an impairment of gas exchange in the lung.

Nominal Atmosphere Concentration Mass Loss, NAC mass loss: The mass loss of material during decomposition per volume of air into which it is dispersed (mg material/liter).

Normobaric Pressure: The barometric pressure of approximately 760 mm Hg. The variation of normobaric pressure may be defined by the range of barometric pressures measured at sea level.

Oxygen Saturation (S_aO_2): The amount of oxygen combined with hemoglobin expressed as a percentage of the oxygen capacity of that hemoglobin.

Oxygen Sea Level Equivalent of Alveolar Air: The oxygen exposure concentration at sea level for which the alveolar partial pressure of O_2 matches the alveolar partial pressure at a given altitude, assuming dry air is inspired.

Oxygen Sea Level Equivalent of Inspired Air: The oxygen exposure concentration at sea level for which the ambient partial pressure of O_2 matches the ambient partial pressure at a given altitude, assuming dry air is inspired.

Oxygen Sea Level Equivalent of Tracheal Air: The oxygen exposure concentration at sea level for which the tracheal partial pressure of O_2 matches the tracheal partial pressure at a given altitude, assuming dry air is inspired.

Plethysmograph: A device for measuring and recording changes in respiratory volume.

Physiological Effects: Effects on the functioning of the body, as opposed to parameters affecting the mind. Thus, a physiological effect of smoke is that it obscures vision, which might have a psychological effect on the willingness of a victim to enter a smoke filled corridor.

Pneumonia (Pneumonitis): Inflammation of the lungs in fire victims due to the direct effects of inhaled chemicals or hot gases or secondarily to skin burns. The initial inflammatory phase may be followed by infection. As it passes through different phases, pneumonia may be life-threatening at any time from one hour after exposure in a fire to several weeks after exposure.

Potency: The toxic potency is a measure of the amount of a toxic substance required to elicit a specific toxic effect. The smaller the amount required, the greater the potency.

Psychological Effects: Psychological effects of exposure to fire scenarios are on the mind of the victim, and may result in a variety of behavioral effects. These are distinct from physiological effects on the body function (see above). A fire victim may suffer both types of effects at various stages of the fire. Interactions between psychological and physiological effects can occur.

Psychomotor: Psychomotor skills are required to perform behavioral tasks involving a series of coordinated movements of the type required to escape from a fire in a compartment (e.g., building, airplane).

Respiratory Minute Volume, RMV: Volume of air breathed each minute (liters/minute). RMV = V_T x f.

Respiratory Rate, f: Respiratory frequency, i.e., number of breaths per minute.

Respiratory Tract: The nose, pharynx, larynx, and large bronchi are termed upper respiratory tract, and the bronchioli, alveolar ducts, and alveoli are termed the lower respiratory tract.

Shock: A reduction in the circulating blood volume with a fall in blood pressure.

Smoke: Total fire effuents, consisting of solid and liquid particles and vapors.

Standard Temperature and Pressure, Dry (STPD): Standard temperature is 0°C and standard pressure is 760 mm Hg, and the air is dry. Gas volumes and partial pressures are expressed under these standard conditions when dealing with metabolic physiology, where the number of molecules of oxygen used and carbon dioxide produced are of interest.

Synergism: Situation where the toxic potency of two or more substances acting in concert is greater than the sum of the potencies of each substance acting alone.

t_{effect}: Exposure time, for a specified concentration of toxic gas, resulting in a toxic effect, e.g., incapacitation or lethality.

$t_{exposure}$: Exposure time, for a specified concentration of toxic gas, resulting in lethality or postexposure lethality of 50% of the animals.

t_i: Time to incapacitation -- The time into an exposure, for a specified concentration of toxic gas, which results in the incapacitation of an animal.

Tenability Limit: Maximum concentration of a toxic fire product or intensity of a physical fire parameter that can be tolerated without causing incapacitation.

Threshold Limit Value, TLV: The time-weighted average concentration for a normal 8- hour workday and 40- hour workweek, to which nearly all workers may be repeatedly exposed, day after day, without adverse effect.

Tidal Volume, V_T: Volume of air exhaled in each breath.

Time of Useful Consciousness, (TUC): The interval that elapses from the start of a low oxygen exposure to the point at which there is a specified degree of performance impairment.

Toxicity: The nature and extent of adverse effects of a substance upon a living organism.

Ventilation, Lung: The volume of air breathed each minute (synonymous with respiratory minute volume).

APPENDIX B
OTHER MODELS

Purser's Model

Purser made the following assumptions on the interactions between narcotic gases[1]:

1. CO and HCN are considered additive.
2. CO_2 increases the rate of uptake of CO and HCN in proportion to its effect on the RMV.
3. The narcotic effect of low oxygen hypoxia is considered to be directly additive to the combined effects of CO and HCN.
4. The narcotic effect of CO_2 is considered to act independently of the effect of the other gasses.

On this basis, Purser predicts a fractional incapacitating dose for narcosis:

$$Total\ F_{IN} = [(F_{ICO} + F_{ICN})\ x\ V_{CO_2} + F_{IO}]\ \underline{or}\ F_{ICO_2}$$

For a one-minute exposure to each gas at a concentration C:

$$F'_{ICO} = \frac{8.2925\ x\ 10^{-4}\ (ppm\ CO)^{1.036}}{30}$$

$$F'_{ICN} = \frac{4.4}{185 - C_{HCN}} \qquad \text{when } 80 \le C_{HCN} \le 180 \text{ ppm}$$

$$= \frac{1}{\exp\ (5.396 - 0.023\ x\ ppm\ HCN)} \qquad \text{when } C_{HCN} > 180 \text{ ppm}$$

$$V_{CO_2} = \frac{exp\ [0.2496\ x\ \%CO_2 + 1.9086]}{6.8}$$

$$F'_{IO} = \frac{1}{exp\ [8.13 - 0.54\ (20.9 - \%O_2)]}$$

$$F'_{ICO_2} = \frac{1}{exp\ [6.1623 - 0.5189\ x\ \%CO_2]}$$

Purser includes both radiant and convective heat in his model. A tenability limit of 0.25W/cm^2 (2.5kW/m^2) for tolerance of radiant heat. A fractional incapacitating dose for convective heat is expressed as:

$$F'_{ICO_2} = \frac{1}{exp\ [5.1849 - 0.0273\ T\ (°C)]}$$

The effects of smoke are included in this model. A tenability limit of extinction coefficient 1.2/m (OD/m = 0.5) is used.

A general tenability limit for severe sensory irritation was set at a concentration of 1mg/L NAC mass loss, and an incapacitating dose for serious postexposure lung inflammation was set at 10 mg/L NAC mass loss for 30 minutes (a CT product of 300 mg min/L).

Narcosis, sensory irritancy, and the effects of heat and visual obscuration are treated separately in this model.

N-GAS MODEL

There are two distinct models, each called the N-Gas Model. Both are based on LC_{50} data obtained by researchers at the Center for Fire Research at the National Institute of Science and Technology (formally National Bureau of Standards) and the Southwest Research Institute. The hypothesis for both models is that while there are hundreds of compounds that can be identified, the effect is caused by only a few (N) key gases. By investigating the effect of exposure to these key gases, singly and in combination, a predictive model can be constructed.

Levin et al. developed a model based on 30-minute LC_{50} data. A six gas model includes the following gases: CO, HCN, CO_2, O_2, HCl, and HBr[86].

$$\textit{Six Gas Model:}\ \frac{m(CO)}{(CO_2) - b} + \frac{(HCN)}{LC_{50\ HCN}} + \frac{21 - (O_2)}{21 - LC_{50\ HCl}} + \frac{(HCl)}{LC_{50\ HCl}} + \frac{(HBr)}{LC_{50\ HBr}} = N\text{-}Gas\ Value$$

where

m	=	-18.	when $(CO_2) \leq 50{,}000$ ppm
b	=	122,000	when $(CO_2) \leq 50{,}000$ ppm
m	=	23	when $(CO_2) > 50{,}000$ ppm
b	=	-38,600	when $(CO_2) > 50{,}000$ ppm

The concentration units are "parts per million" for all gases except O_2 which is percent. Ideally, when this equation is unity, 50 percent of the animals should die.

A seven gas model is used only when NO_2 is present[87].

$$\textit{Seven Gas Model: } \frac{m\ (CO)}{(CO_2) - b} + \frac{21 - (O_2)}{21 - LC_{50\ O_2}} + \left(\frac{(HCN)}{LC_{50\ HCN}} \times \frac{0.4\ (NO_2)}{LC_{50\ NO_2}} \right)$$
$$+ 0.4 \left(\frac{(NO_2)}{LC_{50\ NO_2}} \right) + \frac{(HCl)}{LC_{50\ HCl}} + \frac{(HBr)}{LC_{50\ HBr}} = \textit{N-Gas Value}$$

Bukowski et al.[88] developed a toxicity model which is a part of Hazard I, a fire hazard assessment method for building fires. This toxicity model is based on a curve fit of LC_{50}s vs. exposure times. This model can account for changing gas concentrations with time. Three gases are included in this model: CO, HCN and CO_2. The following linear regressions were developed.

$$(C_{CO} - 1700)\, t = 80{,}000$$
$$C_{HCN} t = 3100$$

where concentrations are in ppm and t is the exposure time for lethality (in minutes) at that concentration.

Bukowski et al. made the observation, based on studies of CO and CO_2 mixtures performed by Levin et al.[52] that "effective toxicity" of CO increases linearly with increasing CO_2 concentration, doubling at a level of 5%. This data was used to produce a CO_2 "correction" to the CO term in the calculation of the FED whereby the denominator is multiplied by the following factor:

$$[100{,}000 - C_{CO_2}/100{,}000]$$

where the CO_2 concentration is in ppm. While the data show this effect diminishing above 5% CO_2, the model holds the correction constant at 5% and above as a conservative assumption. Also note that the data was taken at 30-minute exposure times. A conservative assumption was made that the effect holds for all times.

The resulting equation for the FED, which represents the current N-Gas model in Hazard I (N = 3), is as follows:

$$FED = \sum \left[\frac{C_{CO}\ \Delta t}{C_{CO}\ [80{,}000/(C_{CO} - 1700)]\ [(100{,}000 - C_{CO_2})/100{,}000]} + \frac{C_{HCN}\ \Delta t}{3100} \right]$$

where C_{CO}, C_{CO_2} and C_{HCN} are the average concentrations over the time interval (ppm) and Δt is the length of the time interval (min).

APPENDIX C
BASIC PROGRAM FOR NEW FED_I MODEL

This program should be used with GW-BASIC version D3.20 (Copyright Microsoft, 1986) or later for use on an IBM PC compatible computer. It supports the Hewlett Packard 7550 plotter.

This program computes the FED_I for up to 10 hazards for changing levels of those hazards with time. Temperature and gas concentration are input via keyboard for those times needed to define the hazard - time profile for that test location. Time is input in seconds, and temperature in degrees Celsius.

The program lists the computed individual FED_{GAS} and FED_{HEAT} values for each hazard every second up to the time that $FED_I = 1$. It also lists the computed total FED_{GAS} values for each second of the test.

It will plot three curves on the HP 7550 graphics multipen plotter: FED_{HEAT}, FED_{GASES}, and $FED_{I\ TOTAL}$ as a function of time in seconds.

The program allows the user to do a sensitivity analysis to the assumption that an increase in the V_{CO_2} results in a corresponding increase in the uptake of other gases. The user has the option of setting V_{CO_2} to 1.0, bypassing the automatic calculation of V_{CO_2}.

The program is listed below:

```
5 DIM LAB$(14),LCHK$(14)
10 DIM GAS(12,500), FED(12,500)
12 DIM V(500)
14 FOR J= 1 TO 12
15 FOR I= 1 TO 500
16 GAS(J,I) = 0
17 FED(J,I) = 0
18 NEXT I
19 NEXT J
20 INPUT "NO. OF HAZARDS TO BE INPUT "; NO
23 PRINT "MUST SELRCT CO2 FOR 1ST HAZARD AND TEMPERATURE FOR 4TH"
25 PRINT "  "
30 FOR J=1 TO NO
35 PRINT "TYPE OF HAZARD 1=CO2,2=HCN, 3=HCL, 4=HF, 5=HBR,6=ACROLEIN"
36 INPUT "7=CO, 8=O2, 9=TEMP, 10=NO2  "; GASK(J)
37 PRINT "ENTER % FOR CO, CO2 AND O2 ONLY. ENTER PPM FOR OTHER GASES."
38 PRINT "ENTER DEGREES CELSIUS FOR TEMPERATURE'
39 PRINT "  "
40 INPUT "NO. OF DATA POINTS "; NP
50 FOR K=1 TO NP-1
60 IS=KS
70 Y=X
80 INPUT "CONCENTRATION, TIME (IN SECONDS) "; X, KS
85 GASI(J)=KS
90 GAS(J,KS)=X
100 IF KS=0 THEN GOTO 60
110 IB=KS-IS
120 FOR I = IS+1 TO KS-1
130 GAS(J,I)= GAS(J,IS)+((I-IS)*((X-Y)/IB))
140 NEXT I
150 NEXT K
160 NEXT J
162 PRINT " "
163 INPUT "TYPE 2 FOR RMV CORRECTION. TYPE 0 FOR NO CORRECTION. ";KORR
164 FOR KS = 1 TO 500
165 V(KS) = 1
166 NEXT KS
170 FOR J = 1 TO NO
180 FED (J,0)=0
190 FOR KS=1 TO GASI(1)
200 IF GASK(J)<>7 THEN 205
201 IF GAS(J,KS)*V(KS)<=.01 THEN 204
202 FED(J,KS)=FED(J,KS-1)+(((1/3.425)*V(KS)*GAS(J,KS))/60)
203 GOTO 205
204 FED(J,KS)=FED(J,KS-1)
205 IF GASK(J)<>2 THEN 215
206 IF GAS(J,KS)*V(KS)<=63 THEN 212
210 FED(J,KS)=FED(J,KS-1)+ (((V(KS)*GAS(J,KS)-63)/564 )/60)
211 GOTO 215
212 FED(J,KS)=FED(J,KS- 1)
215 IF GASK(J) <> 10 THEN 220
216 IF GAS(J,KS)*V(KS) <= 290 THEN 219
```

```
217 FED(J,KS)=FED(J,KS-1)+(((V(KS)*GAS(J,KS)-290))/(1.14*10000)) /60)
218 GOTO 220
219 FED(J,KS) = FED(J,KS-1)
220 IF GASK(J) <> 9 THEN 225
221 IF GAS(J,KS) <= 50 THEN 224
222 FED(J,KS)=FED(J,KS-1)+((1/(4.1*100000000#))*(GAS(J,KS)^3.61))/60
223 GOTO 225
224 FED(J,KS) = FED(J,KS-1)
225 IF GASK(J) <>8 THEN 235
226 IF GAS(J,KS) >=11! THEN 232
230 FED(J,KS)= FED(J,KS-1)+(1/(EXP(8.55-.511*(20.9-GAS(J,KS)))))/60
231 GOTO 235
232 FED(J,KS)= FED(J,KS-1)
235 IF GASK(J)  <>6 THEN 243
236 IF GAS(J,KS)*V(KS)<=300 THEN 242
240 FED(J,KS)=FED(J,KS-1)+(1/(1.5+((4*10000)/(V(KS)*GAS(J,KS)+500)))/60)
241 GOTO 243
242 FED(J,KS) = FED(J,KS-1)
243 IF GASK(J) <>3 THEN 250
245 IF GAS(J,KS)*V(KS) <=300 THEN 248
246 FED(J,KS)= FED(J,KS-1)+((1/(3+((3.36*100000!)/(V(KS)*GAS(J,KS)-300))))/60)
247 GOTO 250
248 FED(J,KS) = FED(J,KS-1)
250 IF GASK(J) <> 4 THEN 260
251 IF GAS(J,KS)*V(KS) <= 136 THEN 258
253 FED(J,KS)=FED(J,KS-1)+((1/(3+((1.53*100000!)/(V(KS)*GAS(J,KS)-136))))/60)
256 GOTO 260
258 FED(J,KS)=FED(J,KS-1)
260 IF GASK(J) <> 5 THEN 270
261 IF GAS(J,KS)*V(KS) <= 300 THEN 268
263 FED(J,KS)=FED(J,KS-1)+((1/(3+((3.36*100000!)/(V(KS)*GAS(J,KS)-300))))/60)
266 GOTO 270
268 FED(J,KS) = FED(J,KS-1)
270 IF GASK(J) <> 1 THEN 280
271 IF KORR <>2 THEN 273
272 V(KS)= (EXP(.2496 * GAS(1,KS) + 1.9086))/6.8
273 IF GAS(J,KS)<= 5.5 THEN 278
274 IF GAS(J,KS)<=7 THEN FED(J,KS)=FED(J,KS-1)+ 1/(2193.8-(311.6*GAS(J,KS)))/60
275 IF GAS(J,KS) <= 7 THEN 277
276 FED(J,KS)= FED(J,KS-1)+(1/(EXP(6.1623 -(.5189 * GAS(J,KS)))))/60
277 GOTO 280
278 FED(J,KS)=FED(J,KS-1)
280 NEXT KS
305 NEXT J
400 FOR I= 1 TO GASI(1)
410 FED(12,I)=0
420 FOR J= 1 TO NO
430 FED(12,I)= FED(12,I)+FED(J,I)
440 NEXT J
450 NEXT I
500 CLS
501 REM LPRINT "USE THIS WHEN FED 1,2,3,5,...ARE GASES AND FED4 IS TEMPERATURE"
```

```
502 REM *** LPRINT "                                                    "
503 REM ***Print FEDs on lineprinter: Assume FED1,FED2,and FED3 are Gases***
504 REM ***                           Assume FED4 is temperature        ***
505 REM LPRINT "TIME", "FED HEAT","FED GAS", "FED TOTAL"
510 FOR I=1 TO GASI(1)
511 FED(11,I) = 0
515 FED(11,I) = FED(1,I) + FED(2,I) + FED(3,I) +FED(5,I) + FED(6,I)
516 FED(11,I) = FED(11,I) +FED(7,I) +FED(8,I) +FED(9,I)+FED(10,I)
520 PRINT I, FED(12,I)
521 REM ***  LPRINT I, FED(4,I), FED(11,I), FED(12,I)
523 IF FED(12,I-1) >1 THEN 600
530 NEXT I
600 FOR K= 1 TO NO
610 PRINT "FED";K;"= ";FED(K,I-1)
620 NEXT K
622 PRINT "FED TOTAL = 1 AT ";I-1;"SECONDS"
625 PRINT " "
630 IF KORR = 2 THEN PRINT "FED'S CORRECTED FOR INCREASD UPTAKE (VCO2) "
640 IF KORR <> 2 THEN PRINT "ASSUME VCO2 = 1 "
650 PRINT " "
690 PRINT "TYPE 3 FOR DIFFERENT VCO2 CORRECTION TO THIS DATA. "
700 INPUT "TO STOP TYPE 1, TYPE 2 FOR INDIVIDUAL FED'S, TYPE 0 FOR PLOTTER ";I
710 IF I = 1 THEN 1700
715 IF I=0 THEN 900
718 IF I=3 THEN 162
720 INPUT "FED NO. ";J
725 FOR K= 1 TO GASI(J)
730 PRINT K, FED(J,K)
740 IF FED(J,K) >1 THEN 690
750 NEXT K
760 GOTO 690
900 REM**PLOTTING SUBROUTINE**
920 PRINT "SETUP PLOTTER, PRESS ANY KEY TO CONTINUE"; Z$=INPUT$(1)
930 COLOR 0,7:CLS:LOCATE 12,30: PRINT "PLOTTING IN PROGRESS"
1000 YSCALE = 1.2
1010 INPUT "MAXIMUM TIME FOR X AXIS: CHOOSE 210,240,270,300,330,360,390,420,450,
 OR 480) : ";XSCALE
1020 OPEN "COM2:9600,N,8,1,RS,CS65535,DS,CD" AS #2
1030 PRINT #2,"IN;IP 800 800 9565 6000;"
1040 PRINT #2, "SC 0";XSCALE;" 0 ";YSCALE;";SP1;PU;PA 0";YSCALE;"PD;PA 0 0;PA";
 XSCALE;"0;PU;"
1050 XI = 0
1060 FOR L = 0 TO XSCALE/30
1070 PRINT #2,"PA";XI;" 0;XT;SP1;CP-1.5 -1;LB";XI;CHR$(3)
1080 XI = XI + 30
1090 NEXT L
1100 YI =0
1110 FOR L= 0 TO 6
1120 PRINT #2,"PA 0";YI;";YT;SP1;CP -5.5 -.25;LB";YI;CHR$(3)
1130 YI= YI + YSCALE/6
1140 NEXT L
1150 PRINT #2,"PA ";XSCALE/2;"0;CP -7 -2;LBTime (Seconds)";CHR$(3)
```

```
1160 YLB$ = " Fractional Effective Dose "
1170 PRINT #2,"PA 0";YSCALE/2;"DI 0 1;CP -10 4;LB";YLB$;CHR$(3)
1230 PRINT #2,"SP8;PA 0 "; YSCALE;";YT;"
1280 IL = 1
1285 CO = 2
1290 YS = YSCALE + (YSCALE/5)
1300 REM **ROUTINE TO PLOT FED GASSES (AS FED12) AND TEMP**
1310 FOR I = 1 TO GASI(1)
1312 FED(11,I) = 0
1314 FED(11,I)= FED(1,I)+ FED(2,I) + FED(3,I) + FED(5,I) + FED(6,I)
1316 FED(11,I)= FED(11,I) +FED(7,I) + FED(8,I) + FED(9,I) + FED(10,I)
1318 NEXT I
1350 FOR J = 4 TO 12
1355 IF (J=4) OR (J=11) OR (J=12) GOTO 1360
1356 GOTO 1430
1360 PRINT #2, "SP";CO;";PU;PA 0 ";:PRINT#2, USING"#.#####";FED(J,1);:PRINT #2,"
;PD;"
1370 FOR I = 1 TO GASI(1)
1375 IF FED(J,I)>1.1 THEN 1410
1380 PRINT #2,"PA ";I;" ";:PRINT #2, USING"#.#####"; FED(J,I);:PRINT#2,";"
1390 NEXT I
1410 REM **LEGEND   **
1411 IF J = 4 THEN LAB$(J) = " FED HEAT "
1412 IF J = 11 THEN LAB$(J) = " FED GASES "
1413 IF J = 12 THEN LAB$(J) = " FED TOTAL "
1420 PRINT #2,"SP";CO;"PU;DI 1 0;PA "XSCALE- (2*(XSCALE/6));" ";YS;"PD;PR ";XSCA
LE/12,0;";LB ";LAB$(J);CHR$(3)
1422 CO = CO + 1
1425 YS = YS - .06
1430 NEXT J
1670 CLOSE
1680 GOTO 690
1700 END
```


☆U.S. GOVERNMENT PRINTING OFFICE: 1995 - 604-081/20111